THE REVIEW OF METHODOLOGIES TO EVALUATE THE BENEFITS OF LOWERING THE LEVELS OF LIGHT DUTY VEHICLE EMISSIONS

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FOR

Environmental Protection Service
Environment Canada

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SUMMARY

A review of methodologies to evaluate (i.e., to determine the value, in dollars, of) the expected benefits of reducing emissions levels of light duty vehicles was undertaken on behalf of Environment Canada. Reductions in light duty vehicle emissions may be expected to result in fewer exceedances of National Air Quality Objectives (NAQO) for carbon monoxide CO, nitrogen oxides NOx, and ozone O_3 and a reduction in the deposition rate of nitrates and nitric acid. The resulting benefits would be observed as, e.g., a reduction in the incidence of pulmonary disease, increased agricultural crop yields, fewer days of poor visibility, etc. Establishing dollar values for these disparate measures of benefits would allow the total of the benefits to be expressed in common terms, dollars.

The possible adverse effects of light duty vehicle emissions have been documented in a separate report prepared for Environment Canada (Kolomeychuk et al 1983). The major categories of possible adverse health effects are:

- Adverse effects on human health such as increases in the incidence of: respiratory symptoms (e.g., chest discomfort, cough, headache, sore throat, eye irritation, shortness of breath); aggravation of respiratory disease (e.g., asthma, bronchitis, emphysema); and cardiovascular disease (e.g., increase in the probability of heart attack).

- Changes in forest metabolism, growth and composition; reduced production of forests.

- Reduced yield and value of agricultural crops.

- Contribution of nitric acid to freshwater acidification during spring melt of snowpack containing nitrate.

- Damage to man-made materials (e.g., fading of fabric dyes, yellowing of white fabrics, cracking of springs made of nickel-brass alloy, cracking of tire sidewalls).

- Contribution of NO_2 and nitrate fine particles to reduced visibility.

Due to gaps in the scientific community's knowledge in some cases and a lack of data in others, it was not possible, under the terms of reference of this project, to estimate dollar values for any of the potential benefits, that have been identified, of lowering the levels of light duty vehicle emissions.

No new research or data collection was carried out for this report. It is limited to documenting the state of knowledge on the subject based upon the literature.

The report focuses on the two areas of potential benefits that are closest to quantification: human health and crop damage.

Human Health

The health of all humans is affected to some degree when they are exposed to ambient concentrations of carbon monoxide CO, nitrogen oxides NOx and ozone O_3 that exceed National Air Quality Objectives (NAQO). Exceedances of NAQO are more likely to occur in large cities and, as a result, city residents are more likely to be exposed to elevated concentrations of pollutants than are residents of ~~urban~~ rural areas.

Certain human populations are at greater risk than the population as a whole. The nature and size of populations at risk in four major cities in Canada are summarized in Table S.1.

Table S.1

SIZE OF POPULATIONS AT RISK, 1981

	Population at Risk	City				All Canada
		Edmonton	Montreal	Toronto	Vancouver	
1	General population	657 057	2 828 349	2 998 947	1 268 183	24 343 180
2	Children (under 15)	73 280	279 850	307 230	120 430	5 481 105
3	Population with cardiovascular disease	16 870	63 940	77 560	33 690	606 170
4	Population with acute respiratory disease	22 340	96 160	101 960	43 120	827 668
5	Bronchitics and emphysematics	15 770	67 880	71 970	30 440	584 236
6	Asthmatics	15 770	67 880	71 970	30 440	584 236
7	Anemics	11 830	50 910	53 980	22 830	438 177
8	Pregnant women	6 804	22 115	23 933	11 015	203 678
9	Newly-born infants	12 550	41 860	42 590	19 150	372 451

The incidence of adverse health effects is dependent on the type of pollutant, the sensitivity of the population exposed to it, and the dosage. Dosage is determined by pollutant concentration, rate of ingestion, and duration of exposure. Assuming that appropriate data are available to describe ambient concentrations of pollutants, determining dosage becomes a matter of establishing a relationship between indoor and outdoor concentrations and defining human activity patterns largely in terms of indoor/outdoor exposure.

The primary location of exposure to air pollutants is indoors. Most Europeans, Americans, and most likely most Canadians, spend over 90 percent of their time indoors. The major determinants of indoor air quality are: outdoor pollutant concentrations, indoor pollutant sources and strengths, air exchange rates of structures, and transfer rates of pollutants to sinks.

The GEOMET model (Moschandreas and Stark 1978) appears to be the most suitable of those reviewed for predicting indoor air quality provided adequate time is available to calibrate the model for Canadian conditions and data are compiled to characterize outdoor pollutant concentrations, the housing stock, and indoor pollutant sources.

Models for predicting personal exposure consist of two types: representative individual models; and representative environment models. The former are preferable for estimating cumulative exposure, while the latter require input data that are generally more accessible. Numerical models have not yet been applied extensively to characterize population exposure on an individual basis due to data and economic limitations.

The economic evaluation of health impacts has been approached in two main ways. These can be referred to as:

- output accounting: the value of the output lost and resources foregone because of death, illness, or injury

- welfare accounting: the value of resources individuals would forego (accept) to reduce (increase) a risk of death, illnes, or injury if they had the opportunity.

It is generally considered that welfare accounting is the more theoretically-sound approach of the two. But practical problems limit its usefulness in evaluation of health impacts. The first is that only a small number of empirical studies using a welfare accounting approach have been published. Only one Canadian study using the welfare account methodology has been identified.

The second practical difficulty is that most of the welfare accounting studies deal with mortality, whereas morbidity is the central concern with respect to automotive emissions. Despite the theoretical appeal of welfare accounting, it has been necessary to adopt an output accounting framework to evaluate health effects.

Table S.2 shows some preliminary estimates of the costs of medical treatment associated with individual cases of the diseases that may be caused by exposure to CO, NOx, and ozone. The figures shown in the table include estimates for direct hospital costs (for cases where hospital treatment is required), physicians' costs, prescription drug costs, and an amount for diagnostic services.

The other categories of costs that can be estimated with some accuracy are: the value of lost time in paid employment and the value of lost household work. The aggregate of these costs and medical costs will not be as large as welfare accounting values since they do not include intangibles such as the value of pain and suffering. However, the output accounting estimates can be interpreted as a lower bound on the welfare accounting values.

The next step in quantifying health effects would be to estimate the decline in the number of cases of diseases that may result if the proposed light duty vehicle emission standards are implemented. Two types of data are required to make this estimate. The first is dose-response functions for each adverse effect. Dose-response functions are the mathematical relationship between the dose of a pollutant that an individual receives and the probability that he will experience a particular adverse effect.

The second type of data is estimates of the impact of the proposed light duty vehicle standards on the number, ambient concentrations, and duration of exceedances of National Air Quality Objectives for CO, NOx, and ozone.

The dose-response functions and air quality data required to carry out a quantitative analysis were not available at the time of writing.

Table S.2

ESTIMATED MEDICAL COSTS
($1983)

Substance/Effect	ICD9 Code	Hospital Treatment cost/case		Nonhospital Treatment cost/case	
		Male	Female	Male	Female
Carbon Monoxide					
1. Myocardial infarction (heart attack)	410	1736	1709	63	56
2. Aggravation of angina pectoris (attacks of sharp chest pain due to inadequate blood supply)	413	1400	1385	64	59
3. Intermittent claudication (episodes of leg pain or limping due to inadequate blood supply)	411	1305	1322	58	68
4. Impaired vigilance (visual, auditory, reaction time, time estimation, etc.)	781	1113	1109	45	44
5. Effect on fetal development (decreased birth weight,	319	1650	1768	76	103
	635	na	1070	na	115
increased perinatal deaths	646	na	889	na	36
and decreased weight gain	647	na	1490	na	*
in newborns; reduced learned	648	na	1619	na	72
and general abilities in	653	na	1559	na	102
later childhood)	654	na	1562	na	67

Table S.2 continued

ESTIMATED MEDICAL COSTS
($1983)

Substance/Effect	ICD9 Code	Hospital Treatment Cost/Case		Nonhospital Treatment Cost/Case	
		Male	Female	Male	Female
Nitrogen Dioxide					
6. Increased incidence of acute respiratory illness	460	966	961	34	34
	466	1001	995	41	40
7. Aggravation of respiratory disease (attacks of asthma, bronchitis, or emphysema)	490	1266	1253	42	41
	491	1279	1266	52	46
	492	1317	1305	62	65
	493	1260	1273	87	86
8. Impaired lung function and likelihood of incidence of respiratory symptoms (chest discomfort, cough, shortness of breath)	443	1318	1278	54	47
	478	2023	2014	40	38
Ozone					
9. Increased incidence of discomfort and respiratory symptoms (chest discomfort, cough, headache, sore throat, eye irritation, shortness of breath)	379	1641	1612	47	47
	443	1318	1278	54	47
	478	2023	2014	40	38
10. Aggravation of respiratory disease (same as NO_2)	490	1266	1253	42	41
	491	1279	1266	52	46
	492	1317	1305	62	65
	493	1260	1273	87	86
11. Decreased resistance to infection	460	966	961	34	34
	466	1001	995	41	40
	480	1532	1531	43	38
	487	1004	1009	38	38

na - not applicable

* - no data.

Agricultural Crops

There are two crop-growing areas in Canada where mean, growing-season ozone concentrations are above the threshold for agricultural crop damage: (1) the southwestern portion of Ontario and Quebec along the Windsor to Quebec City corridor; and (2) Vancouver and the lower Fraser River valley.

The extent to which the mean, growing-season ozone concentration would fall if the proposed light duty vehicle emission standards are implemented is not known. It has been estimated that crop losses in Ontario due to ozone damage average about $15 million per year, with a range of $9 million to $23 million. There is no basis for estimating the portion of crop losses in Ontario that can be attributed to light duty vehicle emissions.

The total values of agricultural crops that are at risk of ozone damage in the Fraser River valley and Quebec are: $37 million and $566 million respectively. Estimates of total ozone damage or the amount that can be attributed to light duty vehicle emissions for these regions are not available.

Forests

Losses in forest productivity due to elevated concentrations of ozone are potentially large. However, there are few data available for either ambient ozone concentration in nonurban areas or detailed losses of forest productivity. Therefore, it is not possible to either derive dose-response relationships or identify, with any precision, the areas of forest at risk.

Short-Term Freshwater Acidification

The contribution of nitric acid to short-term freshwater acidification during spring melt of snowpack containing nitrate has been identified in the literature. However, the information required to quantify the effect such as the number and size of freshwater bodies affected, the change in acidity, the types and numbers of organisms at risk, and dose-response functions are not yet available.

Man-Made Materials

While the adverse effects of NO_2 on fabrics--dye fading for a number of fabric and dye combinations and yellowing of some white fabrics--have been well established, the degree to which economic losses occur due to exposure to NO_2 is unclear. The two main problems in quantifying these effects are: the laboratory studies demonstrating the effects are not representative of real-life exposure; and the effects of NO_2 have not yet been successfully separated from the effects of temperature, humidity, and other pollutants.

Evidence exists to show that nitrogen can affect the rate of oxidation of metals and alloys, but a direct relationship between NOx or NO_3 levels in ambient air and corrosion behaviour has not been established. Therefore, quantifying this effect is not possible without further research.

The economic cost of ozone damage to tires is made up mainly of the cost of the antiozonants that are added to sidewall rubber compounds. To the extent that lower levels of light duty vehicle emissions would lead to lower ambient ozone levels and, in turn, to the use of less antiozonants, the dollar value of this effect can be determined. However, it is unlikely that less antiozonants would be used in tires if the proposed light duty vehicle emissions are implemented.

Visibility

Two approaches are generally used for economic valuation of visibility: property value studies and surveys using iterative bidding techniques. The only approach in which it is possible to separate the value of improved visibility from other benefits of reduced air pollution, such as reduced risk to health, is the iterative bidding methodology.

The number of published iterative bidding studies that attempt to evaluate visibility is very small. None is known to have been carried out in Canada, and only about a half dozen such studies have been carried out in various parts of the United States. Although they are not particularly relevant for the situation in Canada, the United States studies indicated that--depending on the area considered, the expected improvement in visibility, and a number of other factors--the willingness-to-pay estimates for individual residents ranged from $43 to $245 per year.

EXAMEN DES MÉTHODES PERMETTANT D'ÉVALUER LES AVANTAGES DE L'ABAISSEMENT DES ÉMISSIONS DES GAZ D'ÉCHAPPEMENT DES VÉHICULES LÉGERS*

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Nous avons examiné, pour le compte d'Environnement Canada, les méthodes permettant d'évaluer les avantages financiers qu'on pourrait retirer de l'abaissement des émissions des gaz d'échappement par les véhicules légers. Cet abaissement permettrait de réduire le nombre de cas où les teneurs ambiantes en monoxyde de carbone (CO), oxydes d'azote (NO_x) et ozone (O_3) sont plus élevées que les Objectifs nationaux de qualité de l'air ambiant et de réduire le dépôt de nitrates et d'acide nitrique. Les avantages prévus pourraient se traduire, par exemple, par une incidence réduite des maladies pulmonaires, un accroissement du rendement des cultures et une diminution du nombre de jours de faible visibilité. L'évaluation financière de ces avantages permettrait de les exprimer globalement par un terme commun, c'est-à-dire en dollars.

Les méfaits possibles des émissions des véhicules légers ont fait l'objet d'un rapport distinct, préparé pour le compte d'Environnement Canada (Kolomeychuk et coll., 1983). Ils peuvent être classés dans les catégories suivantes:

1) effets sur la santé de l'homme tels que l'accroissement de l'incidence de symptômes et malaises (gêne respiratoire, toux, maux de tête, douleurs à la gorge, irritation des yeux, souffle court); l'aggravation des maladies pulmonaires (asthme, bronchite, emphysème, par exemple); et les maladies cardio-vasculaires (accroissement de la probabilité de crise cardiaque);
2) modifications du métabolisme, de la croissance et de la composition des forêts; production forestière réduite;
3) abaissement du rendement des cultures et de la valeur des récoltes;
4) acidification des eaux douces par l'acide nitrique (HNO_3) pendant la fonte printanière du manteau nival renfermant des nitrates;

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* Rapport préparé en vertu du contrat nº KE145-2-0831, passé avec le MAS, en mars 1983.

5) dommage aux matériaux fabriqués par l'homme (par exemple, fanage des colorants des tissus, jaunissement des tissus blancs, craquèlement des ressorts faits d'un alliage de nickel et de laiton, fissuration du flanc des pneus); et

6) réduction de la visibilité par le dioxyde d'azote (NO_2) et les fines particules de nitrates.

À défaut de connaissances scientifiques et de données, il a été impossible, dans le cadre de notre mandat, d'estimer la valeur en argent des avantages potentiels, quels qu'ils soient, qu'il y aurait à diminuer les émissions des véhicules légers.

La recherche et la saisie des données se sont limitées à l'obtention de renseignements sur l'état des connaissances sur le sujet, dans les publications consultables.

Le présent rapport s'est attaché surtout à deux catégories d'avantages qui sont plus facilement quantifiables que d'autres, à savoir ceux qui sont liés à la santé et aux récoltes.

Santé

La santé de tous les humains peut souffrir à un degré ou à un autre de l'exposition à des teneurs ambiantes en CO, NO_x et O_3 qui excèdent les Objectifs nationaux de qualité de l'air ambiant. C'est surtout dans les grandes villes qu'elles dépassent les Objectifs; les citadins sont donc plus exposés que les ruraux.

Certains segments de la population sont plus menacés que l'ensemble de cette dernière. Au tableau S.1 figurent les effectifs et les segments de la population menacés de quatre grandes villes du pays.

L'incidence des effets nocifs pour la santé dépend de la nature du polluant, de la vulnérabilité de la population exposée et de la dose d'exposition, la dose étant déterminée par la teneur en polluant, le rythme d'absorption ainsi que la durée de l'exposition. À supposer qu'on connaisse bien les teneurs ambiantes en polluants, la détermination de la dose d'exposition se réduit à corréler les teneurs dans les locaux et les teneurs à l'extérieur et à répartir grosso modo les formes de l'activité humaine selon le temps passé à l'intérieur et à l'extérieur.

La principale exposition aux polluants atmosphériques a lieu à l'intérieur, où la plupart des Européens, des Américains et probablement des Canadiens passent plus de 90 p. 100 de leur temps. Les principaux facteurs de la qualité de l'air des locaux sont les teneurs en polluants atmosphériques à l'extérieur, les sources de polluants à l'intérieur et leur intensité, le taux d'échange d'air entre les locaux et l'extérieur et la vitesse de transfert des polluants dans les "réservoirs environnementaux".

Le modèle GEOMET semble, parmi tous ceux qu'on a examinés, celui qui permet le mieux de prévoir la qualité de l'air des locaux, à condition qu'on dispose suffisamment de temps pour l'étalonner en fonction des conditions qui existent au Canada et qu'on obtienne des données pour caractériser les teneurs en polluants à l'extérieur, le parc immobilier et les sources de polluants à l'intérieur (Moschandreas et Stark, 1978).

TABLEAU S.1 POPULATIONS VULNÉRABLES, 1981

Population menacée	Ville Edmonton	Montréal	Toronto	Vancouver	Canada
Population totale	657 057	2 828 349	2 998 947	1 268 183	24 343 180
Enfants de moins de 15 ans	73 280	27[illegible] 850	307 230	120 430	5 481 105
Souffrant de troubles cardio-vasculaires	16 870	63 940	77 560	33 690	606 170
Souffrant de maladies pulmonaires aiguës	22 340	96 160	101 960	43 120	827 668
Souffrant de bronchite et d'emphysème	15 770	67 880	71 970	30 440	584 236
Souffrant d'asthme	15 770	67 880	71 970	30 440	584 236
Anémiques	11 830	50 910	53 980	22 830	438 177
Femmes enceintes	6 804	22 115	23 933	11 015	203 678
Nouveau-nés	12 550	41 860	42 590	19 150	372 451

Les modèles permettant de prédire l'exposition des personnes sont de deux types: ceux basés sur des individus représentatifs et les modèles d'ambiances représentatives. Ceux du premier type sont préférables pour estimer l'exposition cumulative, tandis que ceux du second type exigent des données de départ qui sont généralement plus faciles à obtenir. Les modèles numériques n'ont pas encore beaucoup servi à caractériser l'exposition des individus d'une population, à cause de données limitées et de contraintes économiques.

L'évaluation économique des effets sur la santé a été tentée de deux façons principales:

1) comptabilisation des extrants: la valeur des extrants perdus et des ressources inutilisées à cause de la mortalité, de maladies ou de blessures; et

2) comptabilisation du bien-être: la valeur des ressources dont les individus s'abstiendraient (accepteraient) afin de réduire (augmenter) le risque de mortalité, de maladies ou de blessures, s'ils en avaient la possibilité.

La comptabilisation du bien-être est généralement considérée comme la méthode dont la théorie est la plus solide. Toutefois, des difficultés pratiques en limitent l'utilité pour l'évaluation des répercussions sur la santé: la première difficulté est que peu d'études empiriques fondées sur la méthode ont été publiées, et parmi celles-ci, une seule canadienne; la seconde est que la plupart des études de comptabilisation du bien-être se rapportent à la mortalité, tandis que la morbidité est l'élément important lorsqu'il s'agit de gaz d'échappement. Malgré l'attrait théorique de la méthode, il a fallu se rabattre sur la comptabilisation des extrants.

Au tableau S.2, on donne des estimations préliminaires des coûts des traitements médicaux de cas individuels de maladies qui peuvent être causées par l'exposition au CO, aux NO_x et à l'O_3. Les valeurs données comprennent les coûts directs d'hospitalisation (s'il y a lieu), ceux des honoraires médicaux, ceux des médicaments d'ordonnance et ceux des diagnostics.

Parmi les autres catégories de coûts qu'on peut estimer avec une certaine exactitude, mentionnons la valeur du temps perdu par les employés, mais payé, ainsi que la valeur du travail domestique perdu. La somme de ces coûts et des coûts médicaux n'atteint pas les valeurs obtenues par comptabilisation du bien-être car elle ne comprend pas les impondérables tels que la valeur attribuable à la souffrance et à la douleur. Toutefois, les estimations obtenues par comptabilisation des extrants peuvent être considérées comme une limite inférieure des valeurs obtenues par comptabilisation du bien-être.

L'étape suivante de la quantification des effets sur la santé serait d'estimer la diminution de l'incidence des maladies qui peut résulter de la mise en vigueur du projet

TABLEAU S.2 COÛTS MÉDICAUX ESTIMATIFS EN 1983 (en dollars)

Substance et effet	Code ICD9	Coût individuel des traitements hospitaliers		Coût individuel des traitements non-hospitaliers	
		Homme	Femme	Homme	Femme
Monoxyde de carbone (CO)					
1. Infarctus du myocarde (crise cardiaque)	410	1 736	1 709	63	56
2. Aggravation de l'angine de poitrine (douleurs aiguës causées par une irrigation sanguine insuffissante)	413	1 400	1 385	64	59
3. Claudication intermittente (douleurs à la jambe ou boîtement épisodique dus à une irrigation sanguine insuffisante)	411	1 305	1 322	58	68
4. Vigilance perturbée (visuelle, auditive, temps de réaction, estimation de la durée, etc.)	781	1 113	1 109	45	44
5. Effets sur le développement foetal (poids réduit à la					
naissance, accroissement	319	1 650	1 768	76	103
de la mortalité périnatale	635	S.O.	1 070	S.O.	115
et gain réduit du poids	646	S.O.	889	S.O.	36
chez les nouveau-nés;	647	S.O.	1 490	S.O.	*
difficulté d'apprentissage	648	S.O.	1 619	S.O.	72
et diminution des aptitudes	653	S.O.	1 559	S.O.	102
générales dans l'enfance)	654	S.O.	1 562	S.O.	67
Dioxyde d'azote (NO_2)					
6. Incidence accrue des	460	966	961	34	34
maladies pulmonaires	466	1 001	995	41	40
7. Aggravation des maladies	490	1 266	1 253	42	41
pulmonaires (attaques	491	1 279	1 266	52	46
d'asthme, de bronchite	492	1 317	1 305	62	65
ou d'emphysème)	493	1 260	1 273	87	86

Substance et effet	Code ICD9	Coût individuel des traitements hospitaliers		Coût individuel des traitements non-hospitaliers	
		Homme	Femme	Homme	Femme
8. Fonctionnement pulmonaire perturbé et incidence plausible de symptômes respiratoires (gêne respiratoire, toux, souffle court)	443	1 318	1 278	54	47
	478	2 023	2 014	40	38
Ozone (O_3)					
9. Incidence accrue des symptômes d'oppression et des symptômes respiratoires (gêne respiratoire, toux, maux de tête, douleurs à la gorge, irritation des yeux et souffle court)	379	1 641	1 612	47	47
	443	1 318	1 278	54	47
	478	2 023	2 014	40	38
10. Aggravation des maladies pulmonaires (même chose que pour NO_2)	490	1 266	1 253	42	41
	491	1 279	1 266	52	46
	492	1 317	1 305	62	65
11. Résistance réduite à l'infection	493	1 260	1 273	87	86
	460	966	961	34	34
	466	1 001	995	41	40
	480	1 532	1 531	43	38
	487	1 004	1 009	38	38

S.O.: Sans objet
* Aucune donnée

de normes concernant l'émission des gaz d'échappement par les véhicules légers. À cette fin, deux types de données sont nécessaires:

1) pour chaque effet nocif, les fonctions représentant la réponse en fonction de la dose, (c'est-à-dire la relation mathématique entre la dose d'un polluant à laquelle est soumise une personne et la probabilité que cette personne subira un effet nocif donné); et

2) les estimations des répercussions du projet de normes sur le nombre de cas où les teneurs en CO, NO_x et O_3 excèdent les Objectifs nationaux de qualité de l'air ambiant, la valeur de ces teneurs et la durée des épisodes.

Au moment de la rédaction du rapport, les fonctions représentant la relation entre la dose et l'effet ainsi que les données relatives à la qualité de l'air requises pour procéder à une analyse quantitative n'étaient pas disponibles.

Cultures

Au Canada, durant la saison de croissance, deux grandes régions agricoles sont soumises à des teneurs moyennes en ozone, supérieures au seuil où se manifestent des dommages aux cultures: (1) le sud-ouest de l'Ontario et du Québec, dans l'axe Windsor-Québec; (2) la région de Vancouver et la vallée du bas-Fraser.

On ne connaît pas la mesure dans laquelle la teneur moyenne en ozone durant la saison de croissance s'abaissera si les normes proposées sont mises en vigueur. On a estimé que les dommages aux cultures causés par l'ozone, en Ontario, s'élèvent en moyenne à 15 millions de dollars par année, et varient entre 9 et 23 millions de dollars; mais rien ne permet d'estimer quelle proportion est attribuable aux gaz d'échappement des véhicules légers.

La valeur totale des récoltes menacées par l'ozone dans la vallée du Fraser et au Québec est respectivement de 37 et de 566 millions de dollars. On ne possède aucune estimation de l'ensemble des dommages attribuables à l'ozone ou de la proportion des dommages attribuables aux gaz d'échappement des véhicules légers, dans ces régions.

Forêts

Les pertes de la productivité forestière causées par les teneurs élevées en ozone pourraient être élevées. Toutefois, on possède peu de données sur les teneurs en ozone dans les régions non urbaines ou sur les pertes détaillées de la productivité forestière. Il est donc impossible d'en tirer des corrélations entre la réponse et la dose ou de délimiter précisément les surperficies forestières menacées.

Acidification à court terme des eaux douces

On possède des renseignements sur l'effet de l'acidification à court terme des eaux douces par l'acide nitrique au cours de la fonte de la neige qui recèle des nitrates. Toutefois, on n'en possède pas qui permettraient de mesurer les effets (par exemple, le nombre et la superficie des étendues d'eau douce touchées, les modifications de l'acidité, les types et les effectifs des organismes menacés et les relations dose-réponse).

Matériaux fabriqués par l'homme

Les effets nuisibles du NO_2 sur les tissus (fanage des colorants pour un certain nombre de tissus et de combinaisons de couleur et jaunissement de certains tissus blancs) sont bien connus, mais l'ampleur des pertes économiques dues à l'exposition au NO_2 n'est pas bien déterminée. Les deux principales difficultés qu'on rencontre lorsqu'on cherche à mesurer ces effets sont que les expériences en laboratoire ne sont pas représentatives de l'exposition réelle et que les effets dus au NO_2 n'ont pas encore été bien distingués des effets de la température, de l'humidité et d'autres polluants.

Il est avéré que l'azote peut modifier la vitesse d'oxydation des métaux et des alliages, mais une corrélation directe entre les concentrations des NO_x ou de NO_3 dans l'air ambiant et la corrosion n'a pas encore été établie. D'autres recherches sont donc requises pour mesurer cet effet.

Les coûts économiques des dommages causés aux pneus par l'ozone sont surtout constitués par les coûts des agents anti-ozone qu'il faut ajouter aux élastomères du flanc des pneus. On peut déterminer dans quelle mesure la réduction des gaz d'échappement des véhicules légers pourrait se traduire par une réduction des teneurs en ozone de l'air ambiant et par conséquent par une réduction de l'utilisation des agents anti-ozone, ce qui fait qu'on peut en déterminer la valeur en argent. Toutefois, il est peu probable qu'on utilisera moins de ces agents dans les pneus si on met en vigueur les normes proposées.

Visibilité

En général, on utilise deux méthodes d'évaluation économique de la visibilité: l'étude de la valeur des propriétés et les sondages fondés sur la technique des enchères itératives. La seule façon de distinguer la valeur liée à une amélioration de la visibilité des autres avantages d'une réduction de la pollution atmosphérique tels que la réduction des risques pour la santé réside dans les enchères itératives.

Le nombre d'études publiées sur les enchères itératives et dans lesquelles on tente d'évaluer la visibilité est très petit. À notre connaissance, aucune n'a été réalisée au Canada et seules une demi-douzaine ont été réalisées pour diverses régions des États-Unis. Même si elles ne sont pas particulièrement pertinentes pour le Canada, les études américaines montrent que, (selon la région considérée, l'accroissement prévu de la visibilité et un certain nombre d'autres facteurs) chaque résident peut consentir à débourser de 43 à 245 $ par année.

Références

1) Kolomeychuk et coll., "Analyse des incidences socio-économiques de normes proposées limitant les émissions des véhicules automobiles", préparé pour la Direction générale de l'assainissement de l'air, Environnement Canada, 1983.

2) Moschandreas, D.J. et Stark, J.W.C., The GEOMET Indoor-Outdoor Air Pollution Model, EPA-600/7:78-106, 1978.

1. INTRODUCTION

A review of the methodologies to evaluate the expected benefits of reducing emissions levels of light duty vehicles was undertaken on behalf of Environment Canada. The reductions in the levels of hydrocarbons (HC), carbon monoxide (CO), and nitrogen oxides (NOx) emissions from light duty vehicles that were considered are from current maximum allowable levels of:

1.24 HC, 15.5 CO, 1.92 NOx g/km (2.0 HC, 25 CO, 3.1 NOx g/mi)
to proposed maximum allowable levels of:
0.24 HC, 4.35 CO, 0.62 NOx g/km (0.41 HC, 7 CO, 1.0 NOx g/mi)

The impact of the proposed maximum allowable levels on the total amount of emissions from light duty vehicles (i.e., passenger cars and light trucks) in Canada is summarized in Table 1.1.

Table 1.1

ESTIMATED LIGHT DUTY VEHICLE EMISSIONS
AT CURRENT AND PROPOSED STANDARDS
(kilotonnes)

Year	HC			CO			NOx		
	Current	Proposed	Difference	Current	Proposed	Difference	Current	Proposed	Difference
1974	751			7 056			334		
1976	591			6 241			355		
1978	578			6 101			374		
1980	460			4 719			356		
1985	307			3 805			327		
1990	259	209	50	4 036	3 137	899	331	260	71
1995	259	137	122	4 272	2 061	2 211	357	196	161
2000	280	147	133	4 602	2 220	2 382	384	211	173

Source: Air Pollution Control Directorate, Environment Canada

Hydrocarbon emissions have declined steadily since 1974 and are expected to level off at about 260 to 280 kilotonnes during the 1990s if the current standards are maintained. If the proposed emission standards are implemented, light duty vehicle HC emissions are expected to fall to 147 kilotonnes by 2000 or just over half as much as the estimated HC emissions if the current standards are retained.

Carbon monoxide emissions have also declined steadily since 1974, but, instead of levelling off, they are expected to reach a low in 1985 and then increase until the end of the century if the current standards are maintained. By the end of the century light duty vehicle CO emissions will have returned to about the same level as 1980. If the proposed standards are implemented, by 2000 light duty vehicle CO emissions will total 2 220 tonnes, about half of the amount they might otherwise be.

Emissions of oxides of nitrogen have remained fairly constant since 1974 and are expected to remain at about the current level through to 2000 if the current NOx standard for light duty vehicles is maintained. If the proposed emission standards are implemented, light duty vehicle NOx emissions are expected to fall to 211 kilotonnes by 2000, or just over half of the expected amount if the current light duty vehicle standards are maintained.

Estimates of total light duty vehicle emissions, such as those shown in Table 1.1, provide some insight into the potential impact of the proposed emission standards, but, on their own, are not sufficient to quantify the impact. The adverse effects of HC, CO, and NOx emissions and secondary compounds formed from these substances (e.g., ozone and nitrates) on human health, terrestrial ecosystems, aquatic ecosystems, man-made materials, and other populations at risk tend to occur on an episodic basis. That is, on most days the ambient concentrations of CO, NOx, and ozone in most parts of Canada do not exceed the levels at which adverse effects can be observed. National Air Quality Objectives (NAQO) for CO, NOx and ozone are set at a level to provide protection against adverse effects. Therefore, in analysing the potential impact of the proposed light duty vehicle emissions standards, the air quality data that are required are the number of times the NAQO are exceeded, the length of time of each exceedance, and the ambient concentration during each exceedance. The

differences in the number of expected exceedances and their severity (as measured by duration and ambient concentration of the substances) between the status quo scenario (i.e., maintain current standards) and implementation of the proposed standards is the primary measure of the impact of the proposed emission standards.

As the ambient concentration of one or more of the substances climbs above the NAQO, there is a finite probability that some portion of the sensitive population that is exposed will experience an adverse effect. In general, the expected incidence of adverse effects increases as the amount of time exposed and the ambient concentration of the substance (i.e., the dose) increases. Therefore, to calculate the incidence of adverse effects due to NAQO exceedances that occur over a period of time, the mathematical relationship between the dose and the probability of occurrence of an adverse effect (i.e., dose-response functions) is required for each adverse effect and corresponding population at risk.

The final stage in an evaluation (i.e., estimation of the value in dollars) of the benefits of lower levels of light duty vehicle emissions is to convert the estimated reduction in the incidence of adverse effects into dollar terms. This would allow disparate measures of benefits, e.g., reduction in the incidence of pulmonary disease, increased crop yields, fewer days of poor visibility, etc., to be expressed in common-measure dollars.

In summary, the type of data that are required to carry out an evaluation of the benefits of lower levels of light duty vehicle emissions are:

- projections of the differences in the number, durations, and ambient concentration of exceedances of NAQO between the status quo scenario (i.e., maintain existing standards) and a scenario under which the proposed emission standards are implemented;

- dose-response functions for each adverse effect and corresponding population at risk;

- estimates of the size of each population at risk, e.g., the number of people in the general population that have chronic lung disease;

- estimates of the portion of the population at risk that is exposed during exceedances of the NAQO; and

- estimates of the dollar value of one incident of each adverse effect, e.g., the dollar value of one person experiencing an asthma attack.

At the outset of this project there was the expectation that air quality data and dose-response functions as described above would be available from the findings of two related projects that were sponsored by Environment Canada. The appropriate air quality data and dose-response functions were not forthcoming, however. As a result, this report is confined to a description of evaluation methodologies and a compilation of the last 3 types of data described above. For example, it has been possible to estimate the per-incident health-care costs of most of the adverse health effects that are associated with exceedances of NAQO for CO, NOx, and ozone, without regard to the etiology of the effect. But since there is no basis for estimating the expected decrease in the incidence of adverse effects that would result if the proposed light duty vehicle emission standards are implemented, the evaluation could not be carried through to quantitative measurements.

1.1 Study Methodology

Without further research, it is not possible to estimate dollar values for any of the potential benefits of lowering the levels of light duty vehicle emissions that have been identified. Since the terms of reference for the project specified that the work was to be based on a literature search, rather than original research or experimental work, this report is limited to documenting the state of knowledge on the subject.

The report focuses on the two areas of potential benefits that are closest to quantification: human health and crop damage. Protection of human health is the primary motivation for establishing ambient air quality objectives and standards. Of all the potential benefits, reductions in adverse effects on human health are amongst the most complex to evaluate. For these reasons the bulk of the remainder of this report deals with evaluation of health effects. An attempt has been made to provide enough data in the two areas so that when the research gaps are filled--ambient air quality projections and dose-response functions for health effects; ambient air quality projections for crop damage--quantification of the benefits can proceed.

1.2 Adverse Effects of Automotive Emissions

The adverse effects that are considered in this report are those identified by Kolomeychuk et al (1983). The adverse effects and the extent to which a cause-and-effect relationship between ambient concentrations of the substance and the adverse effect has been established are summarized in Table 1.2. Although ambient concentrations at which there is an increased probability of occurrence of each effect are noted in Table 1.2, it is stressed that the numbers do not represent absolute thresholds. In most cases, thresholds are not well established; and some of the effects may occur at lower ambient concentrations than those noted.

Table 1.2

SUMMARY OF POSSIBLE MAJOR EFFECTS OF AUTOMOTIVE EMISSIONS

Substance/Effect	Cause-and-Effect Relationships
HEALTH EFFECTS	
Carbon Monoxide	
1. Myocardial infarction (heart attack)	Increased probability of occurrence when carboxyhaemoglobin (COHb) reaches 3%; minimum ambient concentration of CO: 22.9 mg/m^3 (20 ppm), 8 hours' exposure.
2. Aggravation of angina pectoris (attacks of sharp chest pain due to inadequate blood supply)	Increased probability of occurrence: COHb of 2.5-3%; minimum ambient concentration of CO of 19.5 mg/m^3 (17 ppm), 8 hours' exposure.
3. Intermittent claudication (leg pain or limping due to inadequate blood supply)	Same as #2, above.
4. Decreased work capacity (breathlessness, exhaustion)	Increased probability of occurrence: COHb of >4%; minimum ambient concentration of CO of 32.1 mg/m^3 (28 ppm) 8 hours' exposure.
5. Impaired vigilance (visual, auditory, reaction time, time estimation, etc.)	Increased probability of occurrence: COHb of 3-4%; minimum ambient concentration of CO of 22.9 mg/m^3 (20 ppm), 8 hours' exposure.

Table 1.2, continued

SUMMARY OF POSSIBLE MAJOR EFFECTS
OF AUTOMOTIVE EMISSIONS

Substance/Effect	Cause-and-Effect Relationships
HEALTH EFFECTS	
Carbon Monoxide	
6. Effect on fetal development (decreased birth weight, increased perinatal deaths, and decreased weight gain in newborns; reduced learning and general abilities in later childhood)	Increased probability of occurrence: maternal COHb >4%, fetal COHb of 7%; minimum ambient concentration of CO of 32.1 mg/m^3 (28 ppm), 8 hours' exposure.
Nitrogen Dioxide	
1. Increased incidence of respiratory symptoms (chest discomfort, cough, headache, sore throat, eye irritation, shortness of breath)	Increased probability of occurrence for young children at ambient concentration of NO_2 of 752-1880 $\mu g/m^3$ (0.4-1 ppm); healthy adults at NO_2 of 4700 $\mu g/m^3$ (2.5 ppm).
2. Aggravation of respiratory disease (attacks of asthma, bronchitis or emphysema)	Increased probability of occurrence at ambient concentration of NO_2 of 940 $\mu g/m^3$ (0.5 ppm), although threshold level is not well established.
Ozone	
1. Increased incidence of respiratory symptoms (same as NO_2)	Increased probability of occurrence at ambient concentration of ozone (O_3) of 300-500 $\mu g/m^3$ (0.15-0.25 ppm), or lower.
2. Aggravation of respiratory disease (same as NO_2)	Increased probability of occurrence at ambient concentration of O_3 of 300 $\mu g/m^3$ (0.15 ppm), or lower.

Table 1.2, continued

SUMMARY OF POSSIBLE MAJOR EFFECTS OF AUTOMOTIVE EMISSIONS

Substance/Effect	Cause-and-Effect Relationships
HEALTH EFFECTS	
Ozone	
3. Decreased resistance to infection	Increased probability of occurrence at ambient concentration of O_3 of 360 $\mu g/m^3$ (0.18 ppm).
4. Decreased performance	Increased probability of occurrence at ambient concentration of O_3 of 200-300 $\mu g/m^3$ (0.10-0.15 ppm).
TERRESTRIAL ECOSYSTEMS	
Ozone	
1. Reduced production of forests	Significant effect is observed for sensitive species with ambient concentrations of ozone varying from 60-80 to 200-240 $\mu g/m^3$ (0.03-0.04 to 0.10-0.12 ppm) 24-hour average during growing season.
2. Changes in forest metabolism, growth, and composition	Effects are observed in areas where 1-hour maximum ambient concentration of O_3 reaches 100-800 $\mu g/m^3$ (0.05 to 0.40 ppm).
3. Reduced yield and value of agricultural crops	Significant effects are observed for sensitive plant species at 7-hour midday seasonal average ambient concentration of O_3 of 84-200 $\mu g/m^3$ (0.042-0.100 ppm).

Table 1.2, continued

SUMMARY OF POSSIBLE MAJOR EFFECTS
OF AUTOMOTIVE EMISSIONS

Substance/Effect	Cause-and-Effect Relationships
AQUATIC ECOSYSTEMS	
Oxides of Nitrogen	
1. Contribution of nitrate (NO_3) and nitric acid (HNO_3) to acidic precipitation	Cause-and-effect relationship not well understood. HNO_3 contributes to freshwater acidification during spring melt of snowpack containing nitrate.
MAN-MADE MATERIALS	
Nitrogen Dioxide	
1. Fading of fabric dyes	Significant fading of some fabrics is observed on 12 weeks' exposure at a concentration of 94 $\mu g/m^3$ (0.05 ppm) NO_2 with high humidity (90%) and high temperature (30°C).
2. Yellowing of white fabrics	Effect documented in chamber studies at a concentration of NO_2 of 37.6 $\mu g/m^3$ (0.02 ppm) and exposure of 8 hours.
3. Cracking of springs made of nickel-brass alloy	NO_2 may lower the humidity threshold for corrosion.
Ozone	
1. Cracking of tire sidewalls	Cracking would occur at ambient concentrations if antiozonants were not added to the rubber compound.

Table 1.2, continued

SUMMARY OF POSSIBLE MAJOR EFFECTS OF AUTOMOTIVE EMISSIONS

Substance/Effect	Cause-and-Effect Relationship
	VISIBILITY
Oxides of Nitrogen	
1. NO_2 may cause a brownish haze in a polluted layer when viewed against a relatively clean sky	Color shift should be detectable at a concentration-visual range product of 564 $\mu g/m^3$-km (0.3 ppm-km) NO_2.
2. Nitrate fine particulates contribute to light extinction	Of little consequence generally but may be significant in certain areas.

Source: Kolomeychuk et al (1983)

Only those possible adverse effects of automotive emissions that have been observed, or are suspected, at ambient concentrations of the relevant pollutants experienced in Canada are listed in Table 1.2. There may be other effects that have not yet been observed or for which the scientific evidence is too tenuous to even establish a strong suspicion of causality. That is, it is possible that there are additional potential effects that are currently not known and, for that reason, are not included in Table 1.2. Therefore, the effects listed in the table likely do not represent all of the possible adverse effects of automotive emissions.

1.3 Evaluation of Adverse Effects

1.3.1 Acidic Precipitation

It is not possible under the terms of reference of this study to establish dollar values for the effects of the contribution of automotive emissions to acidic precipitation. Aquatic and terrestrial ecosystems may be expected to suffer adverse effects from acid rain. However, in their review of the literature on the relative importance of sulphur and nitrogen compounds to acidification of surface waters, Kolomeychuk et al (1983) concluded that: "Most of the increase in acidity observed was due to changes noted in sulphate concentrations," and "There is no strong evidence at present for anticipating any appreciable reduction in long-term lake or stream acidification from a reduction in nitrate inputs."

After reviewing the evidence on the effects of acidic precipitation on terrestrial ecosystems, Kolomeychuk et al (1983) concluded: "No documented losses of agricultural crops, forests, or horticultural plants appear to have been clearly identified with acid precipitation in North America."

It must be emphasized that the findings stated above reflect a lack of concrete evidence rather than an evaluation of a large data base that would support a conclusion that there does not exist a cause-and-effect relationship between automotive emissions and long-term, adverse effects caused by acidic precipitation. However, the relationship is not well understood, especially in quantitative terms.

Nitric acid may play a more significant role in short-term acidification of streams and lakes. McLean (1981) reviewed North American and Scandinavian studies and found that nitric acid tends to accumulate in the snowpack over the winter. During rapid melt runoff in the spring, higher than normal amounts of nitric acid are carried into lakes and streams at a time of particular sensitivity in the life cycles of many aquatic species. This phenomenon has become known as "spring shock". McLean's assessment was that nitric acid contributes slightly less than sulphuric acid to observed drops in stream and lake pH (i.e., increased acidity) following snowmelt.

1.3.2 Terrestrial Ecosystems

Losses in forest productivity due to elevated concentrations of ozone are potentially large. However, there are few data available for either ambient O_3 concentration in nonurban areas or detailed losses of forest productivity. Therefore, it is not possible to either derive dose-response relationships or identify, with any precision, the areas of forest at risk. Areas in which ozone may be a problem are generally downwind of and adjacent to cities, while forests that are commercially valuable are, in most cases, long distances from urban areas. Until the phenomenon of long-range transport of pollutants is better understood and data for ambient concentration of O_3 in forested areas are available, it is not possible to determine the value of forest-productivity losses that may be due to elevated levels of ozone.

1.3.3 Man-Made Materials

While the adverse effects of NO_2 on fabrics--dye fading for a number of fabrics and dye combinations and yellowing of white fabrics--have been well established, the degree to which economic losses occur due to exposure to NO_2 is unclear. The two main problems in quantifying these effects are: that the laboratory studies demonstrating the effects are not representative of real-life exposure; and that the effects of NO_2 have not as yet been successfully separated from the effects of temperature, humidity, and other pollutants.

Evidence exists to show that nitrogen can affect the rate of oxidation of metals and alloys, but a direct relationship between NOx or NO_3 levels in ambient air and corrosion behaviour has not been established. Therefore, quantifying this effect is not possible.

The economic cost of ozone damage to tires is made up mainly of the cost of the antiozonants that are added to sidewall rubber compounds. To the extent that lower levels of automotive emissions would lead to lower ambient ozone levels and, in turn, to the use of less antiozonants, the dollar value of ozone damage to tires can be determined. However, rubber compounds are formulated for worst case situations. For tires manufactured in Canada the worst case would be the United States cities with the highest ozone concentrations. Since changes in Canadian automotive emissions levels will have no effect on the worst case situation, Canadian tire manufacturers are unlikely to reduce the amount of antiozonants used in the tires that they produce. Therefore, the economic benefit would be zero.

A final point to note is that, in order for the NOx and O_3-induced damage to man-made materials discussed above to be economically important, the useful life of the products made from the materials must be significantly affected by exposure to the substances. In most instances, useful life is probably determined by normal use rather than pollution damage (EPA, 1978).

1.3.4 Health Effects

The economic benefits of reduced morbidity and mortality of human populations can be calculated using equations of the following form. For adverse effect i, population at risk j, episode where the ambient concentration of a substance is high enough to cause an adverse effect k, city l, and year m, the cost C of excess morbidity or mortality is:

$$C_{i,j,k,l,m} = N_{k,l,m} \times P_i \times S_l \times E_{i,l} \times V_{i,j} \times D_m \quad (1)$$

Where:

- C is cost
- N is the number of times when P_i is expected to be some value greater than zero if the current standards are maintained (base case) minus the number of times when P is expected to be the same value greater than zero if the proposed standards are implemented.
- P is the probability that the adverse effect will occur
- S is the size of the population at risk, i.e., the number of susceptible people.
- E is the percentage of the population at risk that is exposed.
- V is the economic value of the adverse effect.
- D is a discount factor.

The total quantifiable benefit to human populations is the sum of the Cs.

Estimation of value for variables, N and P, will <u>not</u> be addressed in this report as the air quality projections and dose-response functions required to estimate values for N and P, respectively, are not available.

Estimation of values for the remaining independent variables, S, E, and V, is discussed in Chapter 2.

The parameter D is determined from Treasury Board guidelines and is 10 percent for normative purposes and 5 and 15 percent for sensitivity analyses (Treasury Board 1976).

1.3.5 Reduction in Yield of Agricultural Crops

The relationship between reduction in yield of agricultural crops and ambient ozone concentration is, in its simplest form, a linear regression equation of general form;

$$y = a + bx \qquad (2)$$

Where

- y is the predicted percent yield reduction
- a is the predicted yield at a seasonal 7-h/day mean O_3 concertration of 50 $\mu g/m^3$ (0.025 ppm)
- b is a regression coefficient
- x is the 7-h/day mean O_3 concentration

The net benefit of reducing light duty vehicle emissions would be the difference in yield reduction y calculated from projections of the 7-h/day mean O_3 concentration based upon maintaining the current standards and yield reduction y calculated from projections of the 7-h/day mean O_3 concentration based upon implementing the proposed standards.

The economic value of the benefit would be approximately the difference in yield reduction multiplied by the value of the crop harvested in the area being considered.

Projections that relate light duty vehicle emissions to ambient ozone concentrations for crop-growing areas in Canada are not available. Therefore, it is not possible to evaluate the potential impact of the proposed light duty vehicle standards on agricultural crop yields.

The dose-response relationships between ambient ozone concentration and crop reduction have been reasonably well established. Therefore, the increased production of crops that is possible if ambient ozone concentration levels are reduced below threshold levels can be calculated. This was done by Linzon et al (1984) for crops grown in Ontario. While it is not possible to determine the portion of increased crop production that could be attributed to reductions in light duty vehicle emissions, estimates such as those made by Linzon et al (1984) provide an indication of the magnitude of ozone damage to agricultural crops.

The type, quantity, and estimated value of production increases if the ambient ozone concentration is reduced below threshold levels for crops grown in Ontario are presented in Chapter 3.

The type, quantity and total value of agricultural crops grown in areas of Canada, other than Ontario, where ambient ozone concentrations are high enough to cause crop damage are also presented in Chapter 3.

1.3.6 **Visibility**

Two approaches are generally used for economic valuation of visibility: property value studies and surveys using carefully-designed questionnaires (iterative bidding techniques). From property value studies, it is possible to infer the total value that the market places on the benefits of reduced air pollution. However, it is not possible to separate the value of improved visibility from the value of other benefits such as reduced risks to health.

The iterative bidding technique involves a direct determination of economic value from data collected in surveys of populations that are expected to benefit from improved air quality. Simply stated, this technique involves describing alternative levels of air quality to respondents verbally and, if possible, with sets of photographs. The

respondents would then be asked to indicate whether they would be willing to pay a certain price to go from one level of air quality to another level that represents an improvement. The price is varied iteratively until the respondent's willingness to pay for the improvement in air quality is determined.[1]

It is possible, using iterative bidding techniques, to separate the value of visibility from other benefits of improved air quality. However, the iterative bidding technique is not yet well developed, and the studies that have been completed to date must be considered preliminary. Despite their limitations, iterative bidding studies suggest that visibility is of substantial economic value. The value of visibility likely varies greatly from area to area, and there is no basis to assume that the value of visibility determined from an iterative bidding study conducted in Los Angeles, for example, is a good indication of the value of visibility to, for example, residents of Vancouver. However, that is not to suggest that visibility is of little value to residents of Vancouver and residents of other areas of Canada.

The number of published iterative bidding studies that attempt to evaluate visibility is very small. None is known to have been carried out in Canada, and only about a half dozen such studies have been carried out in various parts of the United States.

An excellent review of the state of the art of evaluating visibility that includes a summary of the iterative bidding studies was recently completed and included in the "United States-Canada Memorandum of Intent on Transboundary Air Pollution" documents (MOI 1983). Since no new major work in the field of evaluating visibility has been published since the MOI review was completed, there is little point, in this report, to include a chapter that would either paraphrase or replicate the work done for the MOI. Instead, the review on evaluation of visibility from the MOI documents is included as Appendix 1 of this report.

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[1] For a more rigorous description of the iterative bidding technique see, for example, Brookshire et al (1981) and Appendix 1 of this report.

For the reasons alluded to above--the site-specific nature of visibility evaluation studies, the lack of studies done for Canadian sites, and the early stage of development of iterative bidding techniques--under the terms of reference of this study it was not possible to quantify visibility improvements that may result from reductions in the level of automotive emissions. However, the results of visibility evaluation studies presented in Appendix 1 provide some insight into the dollar value that individuals assign to visibility.

2. HEALTH EFFECTS

2.1 Populations at Risk

The health of all humans is affected to some degree when they are exposed to ambient concentrations of carbon monoxide CO, nitrogen oxides NOx and ozone O_3 that exceed National Air Quality Objectives (NAQO). The specific human populations at greater risk than the population as a whole have been identified (Kolomeychuk et al, 1983). They are:

- people with cardiovascular disease
- asthmatics
- emphysematics
- bronchitics
- pregnant women
- fetuses and new-born infants
- young children

In all cases some percentage of the population at risk, i.e., the most susceptible individuals, can be expected to experience an adverse effect if they are exposed for a sufficient length of time when ambient concentrations exceed the NAQO.

The relationship between the exposure time and concentration (dose) and the probability of the effect occurring--the dose-response function--may vary amongst different age groups, between sex, or because of other factors within each population at risk. For such effects it will require a number of dose-response functions to account for the differences in the susceptibility of various groups. For example, the general population is at risk from increased incidence of respiratory symptoms if it is exposed to ambient concentrations of NO_2 that exceed the NAQO. Young children will experience the adverse effects at lower ambient concentrations and exposure times than healthy adults; and separate dose-response functions for the two groups are required. For most of the adverse effects, the number of dose-response functions and, as a result, detailed definitions

of populations at risk are not known. Therefore, the definitions of the populations at risk used here may have to be modified, and some of the statistics disaggregated to be consistent with the dose-response functions as they become known.

The impact of the proposed light duty vehicle emission standards on air quality will vary from area to area within Canada. The main beneficiaries are expected to be large cities where most of the exceedances of NAQO occur. To account for these differences, population at risk data are required for each city and area that is expected to benefit from lower levels of light duty vehicle emissions. For illustrative purposes, population at risk data are presented for four cities: Edmonton, Montreal, Toronto and Vancouver.

2.1.1 Incidence of Health Disorders

The primary source of data on the incidence of health disorders for Canadians is a survey carried out during 1978 and 1979 by Health and Welfare Canada and Statistics Canada (Health and Welfare Canada, Statistics Canada, 1981). The applicable results from the survey are summarized in Table 2.1.

The survey was based upon self-assessment by respondents and did not include any verification by medical personnel. This may result in the underestimation of some types of heart disease, since an individual may not be aware of its existence.

2.1.2 Pregnant Women and Newly-Born Infants

The size of the population at risk of newly-born infants can be calculated from the rate of live births for each of the cities of interest. They are shown in Table 2.2.

Table 2.1

INCIDENCE OF HEALTH PROBLEMS IN CANADIAN POPULATION[1]
Percentage of Population Affected

	Total Population			Less than 15 Years			15-64 Years			65 Years or Over		
ealth Problem	Total	Males	Females	Total	Males	Females	Total	Males	Females	Total	Males	Females
eart Disease	3.7	3.8	3.6	NA	NA	NA	2.8	3.1	2.6	19.5	20.5	18.7
cute Respiratory	3.4	3.1	3.7	5.8	5.8	5.8	2.8	2.3	3.2	1.6[2]	1.6	1.6
ronchitis & Emphysema	2.4	2.4	2.4	1.3	1.5	1.0	2.4	2.1	2.7	6.3	8.9	4.3
sthma	2.4	2.5	2.2	2.5[2]	3.4	1.6	2.1	1.9	2.3	3.9	5.1	3.0
nemia	1.8	0.5	3.2	0.6	NA	NA	1.9	0.3	3.6	3.8	1.2	5.8

[1] Developed from The Health of Canadians, Report of the Canadian Health Survey, Health and Welfare Canada, Statistics Canada, 1981.

[2] Sampling errors for the estimates in these cells are between 20 and 40 percent. For all other cells, sampling error is less than 20 percent.

NA-Not Applicable

The rate of endogenous production of CO is known to be higher amongst pregnant women. Therefore, all pregnant women, including those whose pregnancies do not result in live births, may be more susceptible to elevated levels of ambient CO. Rates for stillborn children and abortions are also shown in Table 2.2.

Table 2.2

Live-Birth, Stillborn, And Abortion Rates For Selected Provinces

Province	Rate (per 1000 population)		
	Live-Birth	Stillborn[1]	Abortion
Alberta	19.1	.22	3.0
Quebec	14.8	.17	1.4
Ontario	14.2	.16	3.5
British Columbia	15.1	.19	4.6

1. Includes miscarriages

Source: Statistics Canada, "Vital Statistics", Catalogue No. 84-204, 1981.

2.1.3 Smokers

Smokers excrete CO into the air, rather than inhale it from the environment, and are unable to absorb further CO at normal ambient CO levels (EPA 1979). Therefore, smokers must be excluded from the populations at risk due to elevated levels of ambient CO.

Statistics on smoking behaviour of Canadians have been published by Health and Welfare Canada. (Millar 1981). They are summarized in Table 2.3.

2.1.4 The Size of Populations at Risk

Using the 1981 population statistics for the four cities and the data shown on Tables 2.1, 2.2, and 2.3, the size of each population at risk was estimated and is shown in Table 2.4. For illustrative purposes Table 2.4 contains data for 4 major cities as well as for all of Canada.

Table 2.3

INCIDENCE OF SMOKING FOR CANADIAN POPULATION

Percentage of Population Over 15

	Total Population			15-64			65 and Over		
	Male	Female	Total	Male	Female	Total	Male	Female	Total
Prairie Region	34.2	27.1	30.6	36.0	29.9	32.9	19.9	8.8	13.8
Quebec	44.9	33.4	38.9	46.5	36.5	41.1	29.9	12.0	19.5
Ontario	33.1	27.1	30.1	34.9	29.8	32.3	17.5	10.8	13.6
British Columbia	30.5	26.0	28.2	32.8	28.3	30.5	14.9	13.5	14.1

Source: Millar (1981)

Table 2.4

SIZE OF POPULATIONS AT RISK, 1981

	Population at Risk	City				All Canada
		Edmonton	Montreal	Toronto	Vancouver	
1	General population	657 057	2 828 349	2 998 947	1 268 183	23 343 180
2	Children (under 15)	73 280	279 850	307 230	120 430	5 481 105
3	Population with cardiovascular disease	16 870	63 940	77 560	33 690	606 170
4	Population with acute respiratory disease	22 340	96 160	101 960	43 120	827 668
5	Bronchitics and emphysematics	15 770	67 880	71 970	30 440	584 236
6	Asthmatics	15 770	67 880	71 970	30 440	584 236
7	Anemics	11 830	50 910	53 980	22 830	438 177
8	Pregnant women	6 804	22 115	23 933	11 015	203 678
9	Newly-born infants	12 550	41 860	42 590	19 150	372 451

Sources:

1-2 Statistics Canada, "Census Metropolitan Areas and Census Agglomerations with Components," Catalogue No. 95-903, 1981

3 (fraction heart disease; Table 2.1) x (1 - fraction smokers; Table 2.3) x (city population)

4-7 (fraction with disease, Table 2.1) x (city population)

8 (live births x .75 + stillborn x .33 + abortion x .33; Table 2.2) divided by 1000 x (1-fraction smokers; Table 2.3) x (city population)

9 (live births, Table 2.2) divided by 1000 x (city population)

2.1.5 Population Forecasts

Assuming that the first year that the legislation affecting the level of light-duty vehicle emissions will come into effect is 1987, it would be the mid to late 1990s before the entire in-use fleet will be built to the new standards and the full effects of the legislation will be realized. Therefore, it is necessary to use population projections to determine the impact of the reduction in automotive emissions.

The areas under consideration are the Census Metropolitan Areas (CMAs) of Edmonton, Montreal, Toronto, and Vancouver. The CMA is the definition used by Statistics Canada. However, the CMAs are not necessarily consistent with the definiton used by the planners in the respective metropolitan areas. As a result, the metropolitan areas for which population projections are made are not exactly the same as the CMAs.

The relationship between the CMAs and the population forecasts are.

CMA	POPULATION FORECAST
Edmonton	Projection based upon the Edmonton Metropolitan Region which is physically larger than the CMA. For 1981, the CMA represented 91.8 percent of the population included in the region.
Montreal	Projection is based upon the CMA.
Toronto	Projection based upon the Toronto Metro. Region. In 1981, the CMA represented 87.7 percent of the population in the Toronto Metro. Region.
Vancouver	Projection is for the Greater Vancouver Regional District (GVRD) which is slightly smaller than the CMA. In 1981, the GVRD represented 92.2 percent of the population in the CMA.

The level of detail available for each of the forecasts varied widely, both in terms of the population breakdown and the number of years covered by the forecast. The forecasts are shown in Table 2.5.

Table 2.5

POPULATION FORECASTS FOR SELECTED CITIES

Year	Edmonton Metropolitan[1] Region		CMA Montreal[2]	Toronto[3] Metro Region	GVRD[4]
	Low	High			
1986	782 500	800 000			1 254 970
1987				3 738 999	
1990				3 886 265	
1991	850 000	900 000			1 333 461
1995				4 111 890	
1996	929 000	992 500			1 400 959
2000			2 940 000	4 320 353	
2001	1 017 500	1 111 500			1 456 860

Sources:

1 Proposed Edmonton Metropolitan Area Plan, September 1983

2 Discussions with Mr. Fahardy, Montreal Planning Department

3 Unofficial projections from Metropolitan Planning Department, Metropolitan Toronto

4 Greater Vancouver Growth Prospects, Trends and Forecasts, April 1983

These population projections can be used in conjunction with the population profiles (Table 2.6) for each CMA in order to define the population at risk to the level of detail required.

2.1.6 Population Profile

Before attempting to determine the impact of pollutants on the human population, it will be necessary to develop a profile of the population. The profile would highlight factors that are significant to determine the level of exposure to the pollutants and the degree of susceptibility of groups within the populations at risk.

The following four population characteristics should be developed in the population profile:

- Age;
- Sex;
- Occupation; and
- Dwelling Type.

Age is important since children and the elderly have been identified as two of the subgroups within the general population that may be more susceptible to some of the effects.

Disaggregation by sex is necessary since pregnant women are identified as a group at risk and sex may be a factor with certain health problems.

Occupational statistics provide insight into the type of environment in which an individual spends a significant portion of his time. Since air quality is affected by this environment, exposure levels are also affected.

Similarly, dwelling type influences ambient air quality and is an important factor in determining an individual's exposure.

Using the 1981 census data, the population of the four CMAs of interest have been disaggregated by sex, age, occupation, and dwelling type. The data are shown in Table 2.6.

If a quantitative assessment of the health benefits, using a methodology such as the one outlined in Section 1.3.4, was to be carried out, it may be necessary to further disaggregate the populations at risk or to define them differently. This would depend upon the nature of the dose-response functions and the air quality data that would be used in the analysis.

Table 2.6

GENERAL POPULATION CHARACTERISTICS OF THE CMAS

Characteristic	Percentage of Population			
	Edmonton	Montreal	Toronto	Vancouver
Male	50.6	48.6	48.9	49.1
Female	49.4	51.4	51.1	50.9
Total	100.0	100.0	100.0	100.0
Children (under 15)				
Male	11.4	10.1	10.5	9.7
Female	10.9	9.7	10.0	9.3
Adults 15 & over				
Not in the Work Force				
Male 15-64	2.8	4.8	3.2	3.2
Male 65+	2.8	3.6	3.6	4.8
Female 15-64	10.5	14.6	10.5	11.1
Female 65+	3.8	5.6	5.5	6.7
In the Work Force				
Unemployed				
Male	1.2	2.2	1.1	1.4
Female	1.0	2.2	1.1	1.3
By Occupation Environment[1]				
Male				
Office	6.5	6.8	7.7	6.3
Institutional	3.8	3.2	3.3	3.3
Mixed	8.1	8.1	8.6	8.4
Factory	6.0	6.6	6.7	6.4
Outdoor	5.6	2.4	3.1	4.1
Transportation	2.2	1.9	1.6	2.0
Mining	0.4	neg	neg	0.1
Female				
Office	11.2	9.3	12.1	10.5
Institutional	3.8	3.4	3.2	3.6
Mixed	6.8	5.0	6.2	7.0
Factory	0.9	2.1	2.8	1.2
Outdoor	0.3	0.09	0.1	0.1
Transportation	0.2	0.09	0.1	0.1
Mining	0.0	0.09	neg	neg
Total	100.0	100.0	100.0	100.0

Table 2.6 continued

GENERAL POPULATION CHARACTERISTICS OF THE CMAS

Characteristic	Percentage of Population			
	Edmonton	Montreal	Toronto	Vancouver
Total Population by Type of Residence				
Single Detached	56.4	26.9	40.2	58.1
Apartment≥5 Storey	7.9	8.9	29.4	10.0
<5 Storey	21.4	38.4	9.9	22.8
Attached	12.4	13.8	18.8	6.0
Duplex	2.0	12.0	1.4	2.9
Total	100.0	100.0	100.0	100.0

Numbers may not add to 100 percent due to rounding

neg=neglible

[1] The work environments consist of the following Statistics Canada occupation categories:

Office:
- o Managerial, administrative, and related
- o Clerical and related

Institutional:
- o Natural sciences, engineering, and mathematics
- o Religion
- o Teaching and related fields
- o Medicine and health

Mixed:
- o Social science and related fields
- o Artistic, literary, recreational, and related
- o Sales
- o Service
- o Other crafts and equipment operating activities
- o Others unclassified

Factory:
- o Processing
- o Machinery and related
- o Product fabrication, assembling and repairing
- o Material handling and related fields

Outdoors:
- o Farming, agricultural and animal husbandry
- o Fishing, trapping and hunting
- o Forestry and logging operations
- o Construction trades

Transportation:
- o Transport equipment operation

Mining:
- o Mining and quarrying, including oil and gas field operations

All of the figures have been calculated as a percentage of the total population of the CMA.

Source: Statistics Canada, Catalogue #95-903, Census, Metropolitan Areas and Census Agglomeration with Components.

2.2. Exposure to Pollutants

2.2.1 Key Issues

The dose to which populations at risk are exposed may be defined by the ambient (i.e., time-weighted average) concentration of the substance and exposure duration. Before either ambient concentration or exposure duration can be estimated from aggregate air quality data (i.e., data that describes the air quality of entire cities) several issues must be addressed:

1) How does outdoor air pollution affect indoor air quality?
2) Does the effect (if any) vary:
   a) seasonally? and
   b) by type of dwelling?
3) What are current ambient indoor air quality conditions with respect to the compounds under consideration?
4) What are the major determinants of indoor air quality?
5) What is the total daily exposure of individuals to predicted air pollution concentrations?
6) How does exposure vary according to occupation and activity patterns?
7) Are there other major factors influencing personal exposures?

The specificity and precision of the data relating to these questions varied considerably. Attempts have been made to identify major information gaps and to describe limitations and uncertainties associated with the reported research results. Due to the nature of the issues being considered and the extensive air quality literature, a computer literature search was not utilized. Instead, references from recent articles and monographs were examined as a starting point. Researchers active in the field were also contacted; and, from these initial sources, a large number of relevant sources were identified and subsequently examined.

The review is divided into two major sections; the first deals with indoor/outdoor air quality relationships and the second part with personal exposure to air pollution.

2.2.2 Indoor / Outdoor Air Quality Relationships

Automotive emissions are discharged primarily outdoors. Ambient concentrations of emissions can be predicted outdoors through air quality models. These models solely predict outdoor air quality; and, since it is estimated that the average North American urban resident spends less than 2 hours outdoors daily (Szalai 1972), indoor air quality is particularly important in terms of total exposure.

The major objective of this component is to provide a basis for relating changes in indoor air quality to changes in outdoor air quality resulting from modifications to automotive emission standards.

Associated with this objective are matters relating to:

i) current ambient indoor air quality and marginal changes which are likely to occur with the new standards;

ii) exchange rates between indoors and outdoors as a function of building structure, use, and age;

iii) subsequent effects on peak indoor concentrations; and

iv) seasonal variations in exchange rates in relation to heating cycle.

2.2.2.1 Major Determinants of Indoor Air Quality

Air pollutants are bound by the law of conservation of mass; by accounting for all sources and sinks of a given pollutant and the rate of transfer it is possible to predict pollutant concentration (Shair and Heitner 1974, Ishizu 1980). Sources of indoor air pollutants can be divided into two major categories, indoor and outdoor. Indoor sources include such things as fuel combustion for heating and cooking, outgassing of building materials and furniture, and smoking (Lippman 1979, Small 1983).

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Outdoor sources refer to infiltration of outdoor pollutants into buildings. Infiltration and exfiltration of air to and from buildings are influenced by structural design, ventilation systems, induced air pressure differences, meteorology, and human activity (e.g., opening of doors and windows, operation cycle of ventilation systems, etc.) (Kalika et al 1970, Giles et al 1974, Dockery and Spengler 1981, Berglund et al 1982). These variables are correlated partly to the type (commercial, institutional, private residence, etc.) and age of building, its orientation, and geographical location (Spengler et al 1979, Moschandreas et al 1982).

Infiltration rates in most structures are in the order of 1 complete air exchange per hour. Exchange rates are normally measured with all doors and windows closed. As a result, during the seasons when windows are frequently open and in buildings with frequent indoor/outdoor passages, the rates may be much higher (Meyer 1983). With the trend to increasing energy conservation measures in new buildings, exchange rates have been reduced by up to 0.3 air exchanges per hour (Berk et al 1979, Hadley 1981, Small 1983).

Sources of outdoor pollutants include industrial processes, vehicle emissions, and heating fuel combustion. Outdoor concentrations are dependent on proximity to sources, the strength of emissions, and meteorological variables (e.g., wind direction and speed, humidity, etc.) (Dockery and Spengler 1981, Spengler et al 1981).

Sinks of indoor air pollutants may be permanent or temporary (Alzona et al 1979). Permanent sinks include chemical transformation and/or incorporation, settling and adsorption, and exfiltration out of a building (Meyer 1983).

The various combinations of these variables determine indoor air quality for a given building. A number of studies has been undertaken to monitor outdoor/indoor air quality relationships as a first step to understanding the precise quantitative relationships between sources and sinks and resultant indoor air quality (Andersen 1972, Ferris et al 1979, Bellin and Spengler 1980, Spengler et al 1981).

2.2.2.2 Outdoor/Indoor Air Quality Relationships.

Each individual pollutant must be considered independently in reviewing outdoor/indoor air quality since the sources, concentration, reactions, and sinks are often quite different.

Air pollutants can be broken into three general groups; pollutants under consideration have been classified within three categories as follows:

i) chemically-reactive gases
   - O_3
   - NO_2
   - reactive hydrocarbons

ii) chemically-unreactive gases
   - CO
   - NO
   - nonreactive hydrocarbons

iii) suspended particulates
   - particulates

The chemically-unreactive gases are the easiest for which to predict indoor concentrations from outdoor sources. They move from outdoors to indoors without any changes in concentration or chemical form, and the indoor concentration is a direct function of: i) the outdoor concentration, ii) the air exchange rate of the building, iii) indoor source strengths, and iv) the initial indoor concentration (Moschandreas and Stark 1978, Meyer 1983, Wadden and Scheff 1983). This can be expressed mathematically as set out in Section 2.2.2.3 Equation (1).

For the chemically-reactive gases, one must account for the variables described above plus the reaction rate of the pollutant. This is normally dealt with through a decay constant as shown in Equation (3) (Shair and Heitner 1974, Moschandreas and Stark 1978, Lippman 1979).

Another term must be introduced to deal with suspended particles as described by Alzona et al (1979). Particulates are filtered out of the air as they move through walls and cracks of buildings and, therefore, a direct reduction in concentration occurs. Settling rates of particles indoors can be treated with a decay constant as mentioned above; however, another term is required to describe the resuspension of particles by human activity and air turbulence. The resuspension constant is a function of air circulation, activity patterns, and particle sizes.

2.2.2.3 **Mass Balance Models**

The generalized theoretical models for predicting indoor air quality are based on the law of conservation of mass and the chemical and physical transformations that can reduce pollutant concentrations. A variety of models and algorithms has been developed (Shair and Heitner 1974, Ishizu 1980, Berk et al 1981, Dockery and Spengler 1981, Wadden and Scheff 1983) and the GEOMET model developed by Moschandreas and Stark (1978) is perhaps most suited to the needs of this analysis.

The operational algorithm for the GEOMET model is presented first for non-reactive gases. In subsequent forms, each of the other classes of pollutants is treated.

Equation (1)

$$[P_n\ (t_i)] = ([P_n\ (t_{i-1})] - m_i * t_{i-1} - (b_i + S_i/(V*x_i) - m_i/x_i)) * e^{-x_i*(t_i - t_i)} + (b_i + S_i/(V*x_i) - m_i/x_i) + m_i t_i$$

where

$[P_n\ (t_i)]$ = the indoor pollutant (P_n) concentration at time t_i.
$[P_o\ (t_i)]$ = the outdoor pollutant concentration at time t
$m_i = ([P_o\ (t_i)] - [P_o\ (t_{i-1})]\ /\ (t_i - t_{i-1})$
(average rate of change of outdoor pollutant concentration over time interval t_{i-1} to t_i)

$b_i = [P_o(t_i)] - m_i t_i$
(average outdoor pollutant concentration over time interval t_{i-1} to t_i)

S_i = internal source pollutant generation rate over time interval t_{i-1} to t_i.

x_i = air exchange rate over time interval t_{i-1} to t_i

V = volume of building

When the outdoor pollutant concentration is constant, this equation simplifies to the following form:

Equation (2)

$$[P_i] = ([P_{i-1}] - [P_o(t_i)] - S_i/V*x_i) * e^{-(t_i - t_{i-1})} + [P_o(t_i)] + S_i/V*x_i$$

The first term in the equation deals with the time lag for an equilibrium to be reached inside the building, and has a typical diffusion equilibrium form. The second two terms specify the outdoor and indoor concentrations respectively.

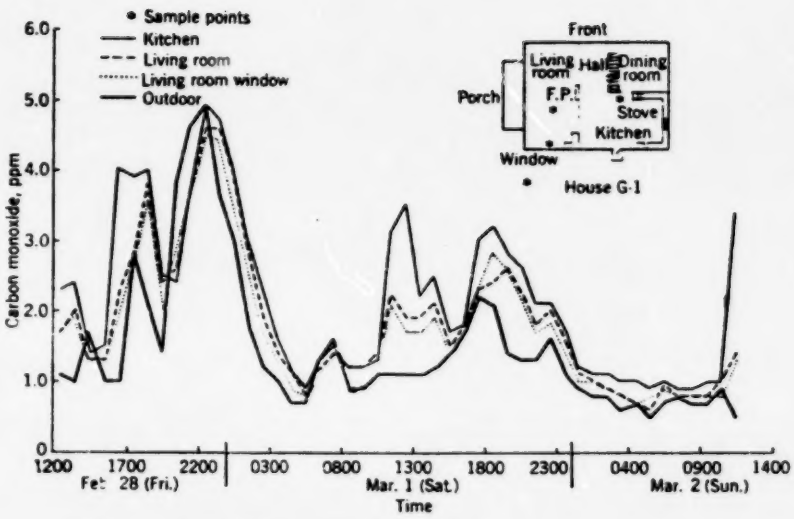
The response of this equation is similar to observed concentrations for nonreactive pollutants particularly for carbon monoxide (Moschandreas and Stark 1978, Ott and Flachsbart 1982).

Figure 1 is reproduced from Yocom et al (1971) and shows the time lag between the peak in outdoor concentration of CO and the resultant lagged indoor response. The lag as shown in Equation (2) is dependent on the air exchange rate of the building; the lag increases as the exchange rate decreases.

The net effect of the time lag is to dampen indoors the extreme concentrations in outdoor pollutant concentration; however, the average concentration both outdoors and indoors is identical if no internal sources of the pollutant are present.

The equation for reactive gases is similar to Equation (1) except that one must include a term D_i, the decay factor over time interval t_{i-1} to t_i.

Figure 1



Time variation of carbon monoxide for house G-1.

Source: Yocom, Clink and Cote (1971)

Equation (3)

$$[P_i] = ([P_{i-1}] - m_i*(x_i/(D_i+x_i)*t_{i-1} - (1/(D_i+x_i) * (x_i*b_i + S_i/V - (m_i*x_i/(D_i+x_i)))) * e^{-(x_i+D_i)*(t_i-t_i)} + (1/D_i+x_i) * (x_i*b_i + S_i/V - (m_i*x_i/(D_i+x_i)) + m_i*(x_i/(D_i+x_i))*t_i$$

Equation (3) behaves similarly to Equation (1) except that indoor average concentrations tend to be lower when $S_i = 0$. In effect, the average is directly proportional to D_i. If D_i is large (i.e., the pollutant rapidly breaks down indoors), then the average indoor concentrations will be relatively low compared to average outdoor concentrations. The reverse is true if D_i is small. Each pollutant has its own decay factor value; decay values are shown in Table 2.7 for some of the gaseous pollutants under consideration in this study. Values are not available for reactive hydrocarbons, and each compound would have a unique decay factor value depending on chemical reaction and settling rates. These values would need to be derived independently from empirical data.

The final modification required for the general GEOMET algorithm is to include settling and resuspension coefficients for suspended particles. This could be dealt with through an aggregated decay factor or it could be entered in an explicit way as described by Alzona et al (1979). Such a modification to the GEOMET model has not been reported in the literature, and it is outside the scope of this report to develop this mathematical form of the equation. Accordingly, the GEOMET model in its current form is not capable of predicting particulate concentrations indoors.

2.2.2.4 Empirical Models.

All models are derived ultimately from observations (i.e., empirical data). The difference between empirical and mass balance models is in the model formulation procedure. Empirical models are derived by attempting some type of multivariate statistical analysis (frequently least squares regression) on a data set to identify significant variables and their associated coefficients (Wadden and

Table 2.7 Decay Factors for the GEOMET Indoor Air Quality Model

Pollutant	Decay Factor (per hour)
CO	0.00
NO	0.00
NO_2	1.39
O_3	34.66
Non-reactive Hydrocarbons	0.00
Reactive Hydrocarbons	not available

Source: Moschandreas and Stark (1978)

Scheff 1983). Mass balance models are based on theoretical relationships. Then each component in the model is calibrated using recorded data, and the entire model is validated against a set of independent observations. Empirical models are often referred to as "black box" models, and they may have a high level of predictive accuracy under specific conditions. However, they lack the adaptability and generality of mass balance models.

The most pertinent empirical model to this analysis is one developed by Pengelly et al (1983). In this study, a total of 16 schools and 20 homes in Hamilton, Ontario, were monitored in terms of outdoor and indoor air quality. Their data base included information on season, age of school, presence of gas stoves and smokers, and the pollutants, NO_2, SO_2, and suspended particulate material (SPM). The relationships that they observed were quite consistent with much of the literature. Namely, where indoor sources were negligible, the indoor/outdoor (I/O) ratios for NO_2 were 0.75 to 0.80.

In contrast, SPM I/O ratios were from 0.90 to 1.34, and there was poor correlation between indoor and outdoor concentrations; this likely reflects the relative unimportance of outdoor sources to indoor SPM concentration and suggests the presence of significant internal sources. The presence of smokers did account for a significant amount of the observed variation, and they estimated the presence of smokers added an average 35 μ/m^3 of particulates. The major indoor sources of NO_2 were gas stoves.

The regression equations developed are broken out by schools and homes as shown.

Equation (4): Schools/Nitrogen Dioxide

$$[P_i(NO_2)] = -1.0 + .8 * [P_o(NO_2)]$$

Equation (5): Homes/Nitrogen Dioxide

$$[P_i(NO_2)] = -0.5 + 17.6(Gas) + 0.86 * [P_o(NO_2)]$$

Equation (6): Homes/Particulates (Heating Season Only)

$$[P_i(SPM)] = -24 + 1.13 * [P_o(SPM)] + 35(Smoker)$$

where P_i = the indoor pollutant concentration
P_o = the outdoor pollutant concentration
NO_2 = nitrogen dioxide
SPM = suspended particulate material
Gas = the presence of gas stoves in the buildings
Smokers = the presence of smokers in the buildings

Equation (6) is valid only for the heating season. The last two variables, gas stoves and smokers, take values of 0 or 1 indicating absence or presence. In more sophisticated analyses, specific values for smoking and cooking frequency and emission strength are input (Spengler et al 1979 and 1981).

This empirical equation does not provide the opportunity for dealing with temporal variations in pollutant concentrations of less than approximately 48 hours since the input data represents long-term averages. Exposure estimates must, therefore, be cumulative and based on average indoor concentrations or the time lag resulting from equilibration of outdoor and indoor concentrations must be assumed to be 0. The acceptability of this assumption will depend on the precision required for exposure estimates and the inputs available for other variables in the overall exposure-dose-response equations (e.g., predicted outdoor concentrations, activity patterns, structural characteristics of buildings, etc.).

2.2.3 Personal Activity Patterns And Exposure Rates

2.2.3.1 Objectives

In the preceding section the relationship between outdoor and indoor air quality was reviewed and methods to predict indoor air quality were presented. The overall objective, however, is to predict changes in mortality and morbidity due to air pollution. Mortality and morbidity rates are dependent on the type of pollutant, dosage, and the sensitivity of the receptor. Dosage is determined by pollutant concentration, rate of ingestion, and duration of exposure. Exposure in turn is a function of the temporal and spatial distribution of air pollutant concentrations and the coincident distribution of humans. Rate of ingestion is proportional to ventilation rate, which is dependent on the specific activity being performed by an individual (Lee and Mage 1979).

The objectives of this section are to:

i) Characterize activity patterns of urban populations in relation to air pollution patterns; and

ii) Review methods to estimate personal exposure rates to air pollutants.

2.2.3.2 Activity Patterns

Considerable work has been done characterizing activity patterns and time budgets, particularly for urban populations (Szalai 1972, Tomlinson et al 1973, Chapin 1974, Robinson 1977). Unfortunately much of the work is focussed on American and European populations. Chapin (1974) reported on time budget surveys for Washington, D.C. The results are disaggregated by workdays and weekends and by hour and activity (Figures 2a, 2b, 2c). Several general trends are constant among the various reported studies and these are summarized graphically in Figure 3.

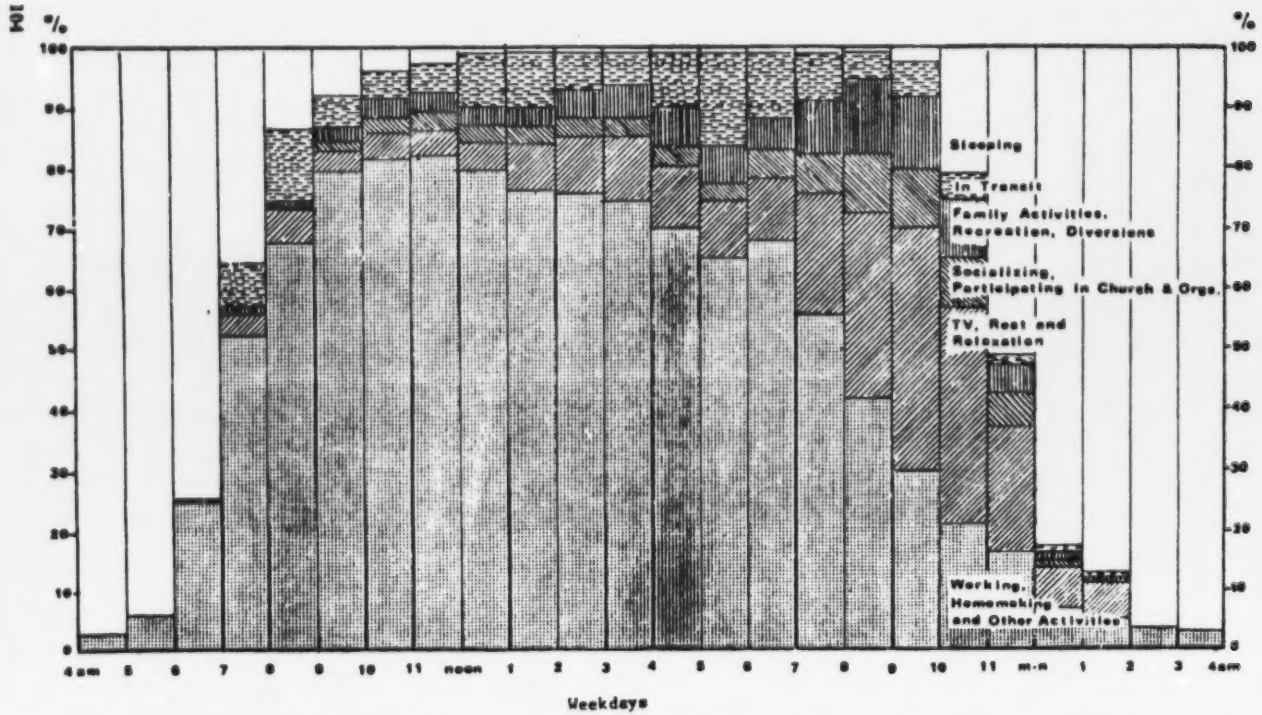


Figure 2a. What heads of households and spouses are doing different hours of a 24-hour period on an average weekday, Saturday, and Sunday—Washington, spring 1968.

Source: Chapin (1974)

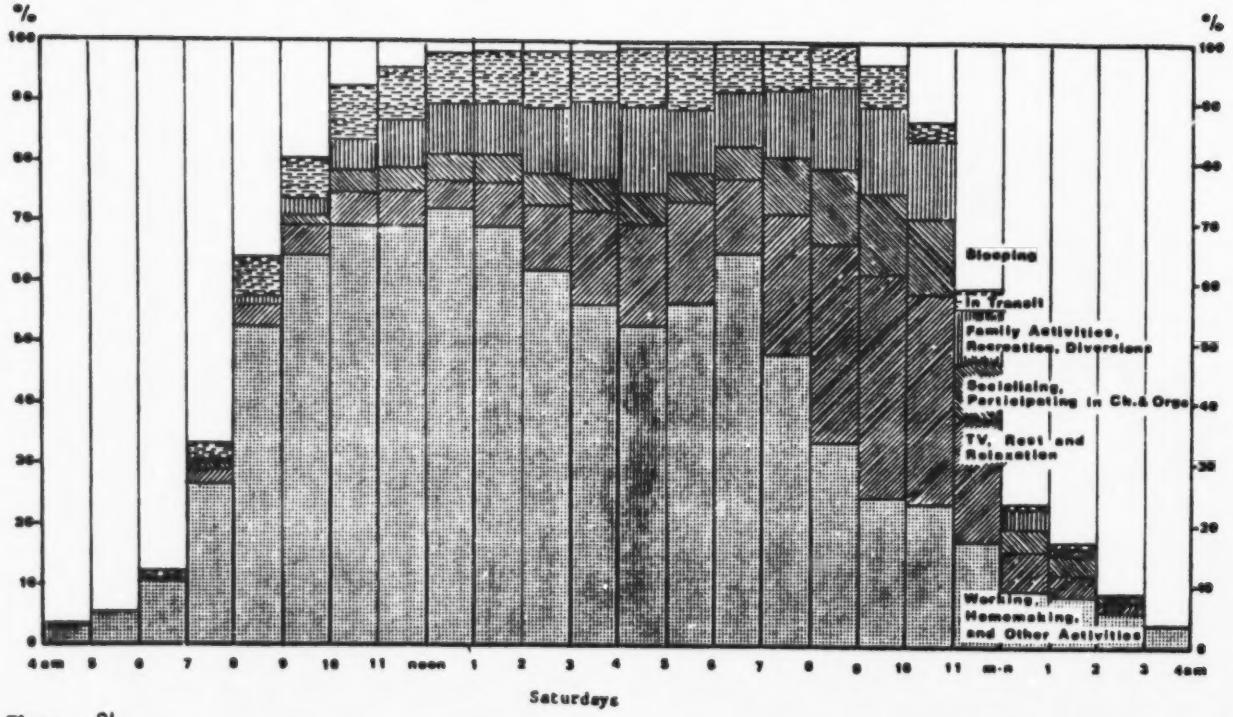


Figure 2b

Source: Chapin (1974)

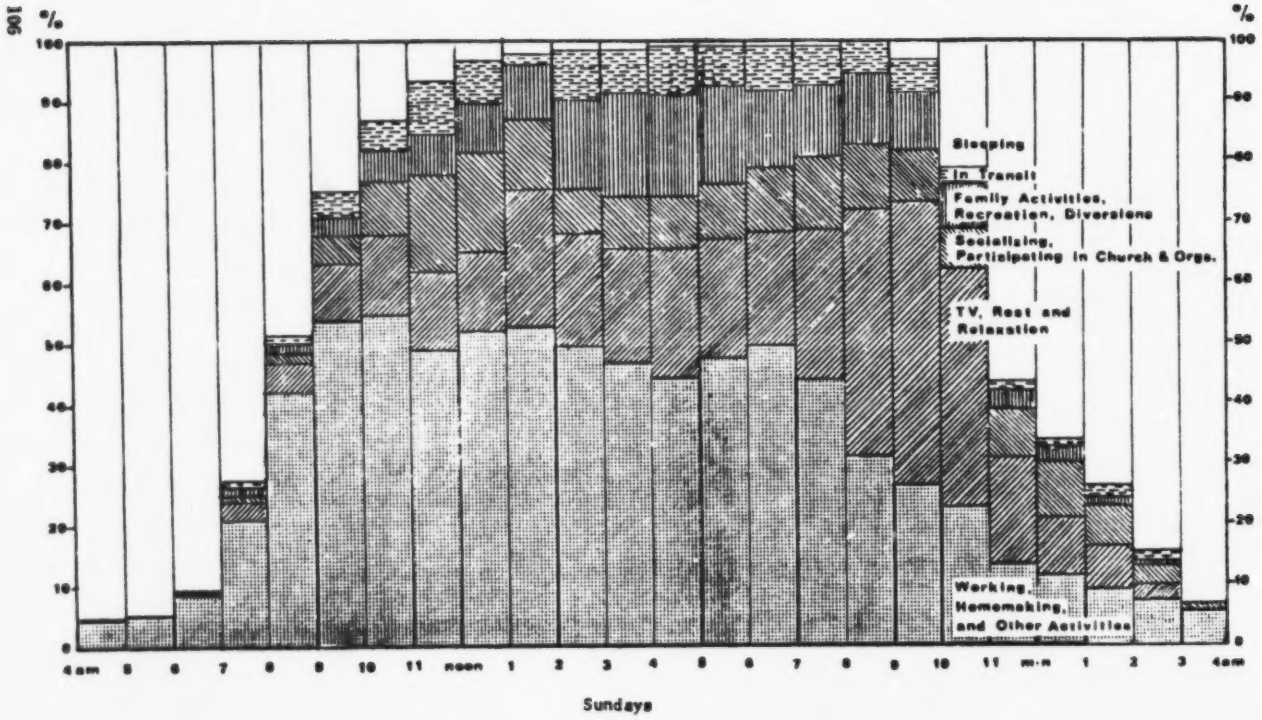
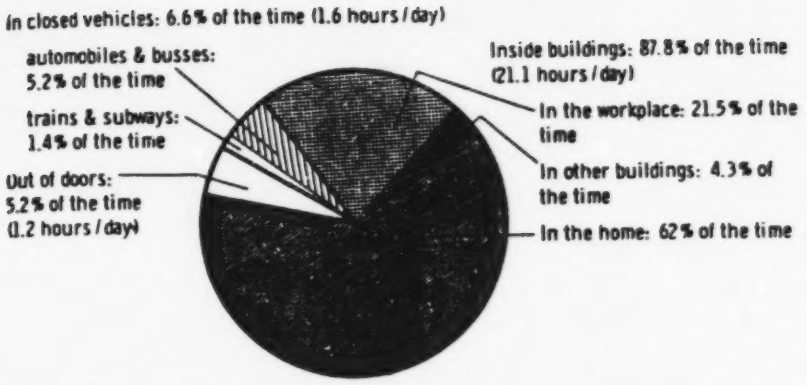


Figure 2c

Source: Chapin (1974)

Figure 3


In closed vehicles: 6.6% of the time (1.6 hours/day)
automobiles & busses: 5.2% of the time
trains & subways: 1.4% of the time
Out of doors: 5.2% of the time (1.2 hours/day)
Inside buildings: 87.8% of the time (21.1 hours/day)
In the workplace: 21.5% of the time
In other buildings: 4.3% of the time
In the home: 62% of the time

Time budget — average day. Location of activities — 38 foremen, supervisors, and managers, ages 40 to 50 years. New York State, 1977.

Source: Hinkle (1980)

By far the greatest amount of time is spent in the home (i.e., 14.5 hours of the day); however, on the average, about 7.5 hours of that time are spent resting during which ventilation rates are the lowest and, therefore, ingestion rates are reduced. The next most time is spent in the work place (i.e., 5 hours). The quality of the air in the workplace can be predicted as for private residences with consideration given to internal sources of pollutants. Approximately 1.6 hours per day are spent in closed vehicles. Exposure in this environment is dependent primarily on type and condition of vehicle, traffic volume, and speed (Brice and Roessler 1966, Cortese and Spengler 1976, Jaeger 1981, Meyer 1983). Finally, on the average, 1.2 hours are spent out of doors. Often outside activities (e.g., sports and physical activity) are associated with increased ventilation rates (i.e., twice average rates) so that the effective dosage outdoors may be proportionately greater than the percentage of time estimate reflects. Taking this fact into consideration, however, will not significantly affect the dominance of indoor air quality in determining cumulative exposure rates.

Generalized estimates of the human population distribution are possible using these studies provided that data for location of residence and work place can be aggregated to generate representative activity patterns of given population groups. The existing data base is not adequate, however, to provide mor detailed breakdowns for individual receptor sensitivity categories. Most of the activity pattern and time budget research has been focussed at a broad scale and for sociological, as opposed to epidemiological, purposes.

2.2.3.3 Personal Exposure

Basic Concepts

Personal exposure may be measured on both incident and cumulative bases (Bates et al 1970, Lee and Mage 1979). The best measure depends on the pollutant and whether its effects are short term and reversible or long term and chronic. Despite which measure is used, an individual's exposure will be a function of activity pattern and the air quality at the time(s) and location(s) activities take place.

Incident exposure is perhaps easier to estimate. Threshold pollutant concentrations for detectable significant effects need first be established. Outdoor and indoor air quality models can be used to predict concentrations. All sites exceeding these threshold limits can then be analysed with respect to the population exposed. That segment of the population present during the period of the exceedance can be estimated based on site-use characteristics.

Cumulative exposures are more difficult to estimate. Cumulative exposure is normally expressed as time-weighted average concentrations (Moschandreas and Morse 1979, Duan 1982).

Equation (7)

$$CE = [E_i]*t_i$$

where CE = the cumulative exposure over time T;
$[E_i]$ = the pollutant concentration at site i;
t_i = the number of hours (minutes) spent at site i; and
$T = t_i$

To estimate cumulative exposure, the daily (hourly, weekly, annual, etc.) activity patterns of individuals must be matched with predictions of air quality. Equation (7) was suggested by Duan (1982) to estimate total dosage of an individual to a given air pollutant; however, the general equation could be improved by relating ventilation rate to a given site; such a modification is shown in Equation (8).

Equation (8)

$$CE = VR_i *[E_i] * t_i$$

where VR_i = the average ventilation rate (l/min) at site i.

By calculating cumulative exposures in this way, comparisons between sites can be made directly on the basis of dosage as opposed to ambient concentrations.

Modelling

Modelling of human dosage can be undertaken using two basic approaches (Duan 1982, Meyer 1983). The first option (termed representative individual models) is to generate hypothetical individuals that are representative (in terms of location and type of activities and pollutant sensitivity) of specific groups in the population and to model each individual in terms of activities and resultant incident or cumulative dosage. These results are then generalized on the basis of the size of the population which each representative individual characterizes.

An alternative approach (termed representative environment models) is to select representative environments to which the population is exposed. Then, by combining general population activity patterns with air quality predictions for each site (i.e., environment), exposure rates can be calculated as a percentage of the population, exposure duration, and pollutant concentration (Moschandreas and Morse 1979).

Both of these approaches have certain strengths and limitations. The data requirements for the first could be impractical if the number of representative individuals is large. At the same time, however, it permits efforts to be focussed on sensitive at-risk groups and yields integrated cumulative exposure directly.

The second approach requires less input data provided that the number of environments is limited. In addition, much of the input data can be developed fairly easily from reported sources. The major limitation relates to predicting individual cumulative exposures; at each site, a given percentage of the population will be exposed, but there is not a direct relationship between the fraction of the population exposed at each site and the cumulative exposure of an individual within a given population group.

For example, 10% of a given population group may be exposed to high pollutant concentrations at two different sites. If a high degree of correlation exists between the people comprising the 10% exposed at one site and those exposed at another, then many individuals in the group will not have been exposed while a smaller fraction will receive a much higher exposure than expected. For many

pollutants, a threshold dosage is defined below which no effect occurs. It is possible that the average exposure rate of a population group could be below a given threshold, and it would be concluded that no negative health effect would occur. However, in actual fact, that segment of the population group which was exposed to both sites may exceed the threshold dosage and experience negative health effects. This type of predictive error is reduced somewhat, but not eliminated, with the representative individual model approach.

Representative Individual Models

These models can be constructed on a theoretical component basis or from empirical data. In the first case, representative population groups must be selected, and then for each group a representative activity pattern is constructed. Activity patterns are combined with compatible predictive air quality model results. Incident or cumulative exposure rates may then be predicted.

Empirical models are derived from personal exposure monitoring (PEM) data. In these studies, research subjects carry monitoring devices throughout their daily routines, and air pollutant concentrations are automatically recorded (Cortese and Spengler 1976, Ott and Flachsbart 1982, Silverman et al 1982). These records provide a direct measure of incident and cumulative exposure and can be related on the basis of activity pattern records to exposure rates in various environments (Lee and Mage 1979). Provided sufficient data of this sort could be obtained for each representative individual type, predictions of exposure can be carried out using statistical analysis procedures.

A number of PEM studies have been undertaken (e.g., Berk et al 1981, Meyer 1983). A joint study is currently underway in Toronto and Hamilton (Silverman et al 1982), but the potential level of disaggregation among groups and environments is quite limited due to the relatively small sample size (i.e., 26 individuals). These studies have effectively demonstrated, however, that significant discrepancies exist between exposure levels predicted from stationary monitoring devices and PEM devices. For example, in San Francisco commercial buildings, Ott and Flachsbart (1982) found average CO concentrations of 3.02 ppm using PEM

compared to 2.00 ppm reported for fixed monitoring stations. They concluded that "the CO concentrations in commercial settings are above the values measured by nearby fixed air monitoring stations and have little relationship to fixed station values.". In another study in Boston by Cortese and Spengler (1976), it was concluded that "[measurements] at six fixed monitoring stations ... underestimated mean 1-hour personal exposure [to CO] by a factor of 1.3 to 2.1.". They did find, however, that "[fixed] location measurements were representative of 8-hour average population exposure".

Representative Environment Models

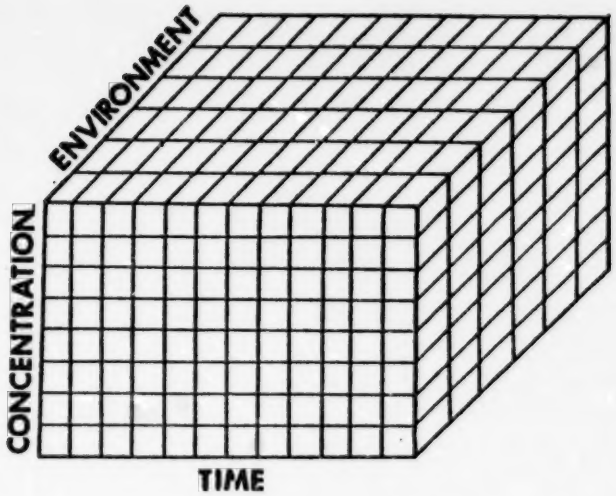
Personal exposure predictions using this method have been discussed fairly extensively in the literature (Moschandreas and Morse 1979, Meyer 1983, Wadden and Scheff 1983, Berk et al 1981). Figure 4 conceptually depicts the basic approach. Within each cell of the three-dimensional matrix, exposures of population groups are predicted on the basis of general activity patterns and pollutant concentrations (Figure 5). Air quality must be input such that the time, environment, and concentration rows can be completed across the matrix.

The size of the matrix is clearly a function of the number of categories in each component. For example, Moschandreas and Morse (1979) used hourly time intervals, 10 concentration divisions, 5 environment types, and 4 population groups yielding 4,800 potential cells. While not all of these cells are filled, the size of the population groups and environment types were quite broad, and each easily could have been increased to 3 or 4 times as many categories. Representative individual models also would require this number of cells to achieve the same level of precision.

Both types of personal exposure models are compatible with the two types of outdoor/indoor air quality models described in Section 2.2.2.3.

Figure 4

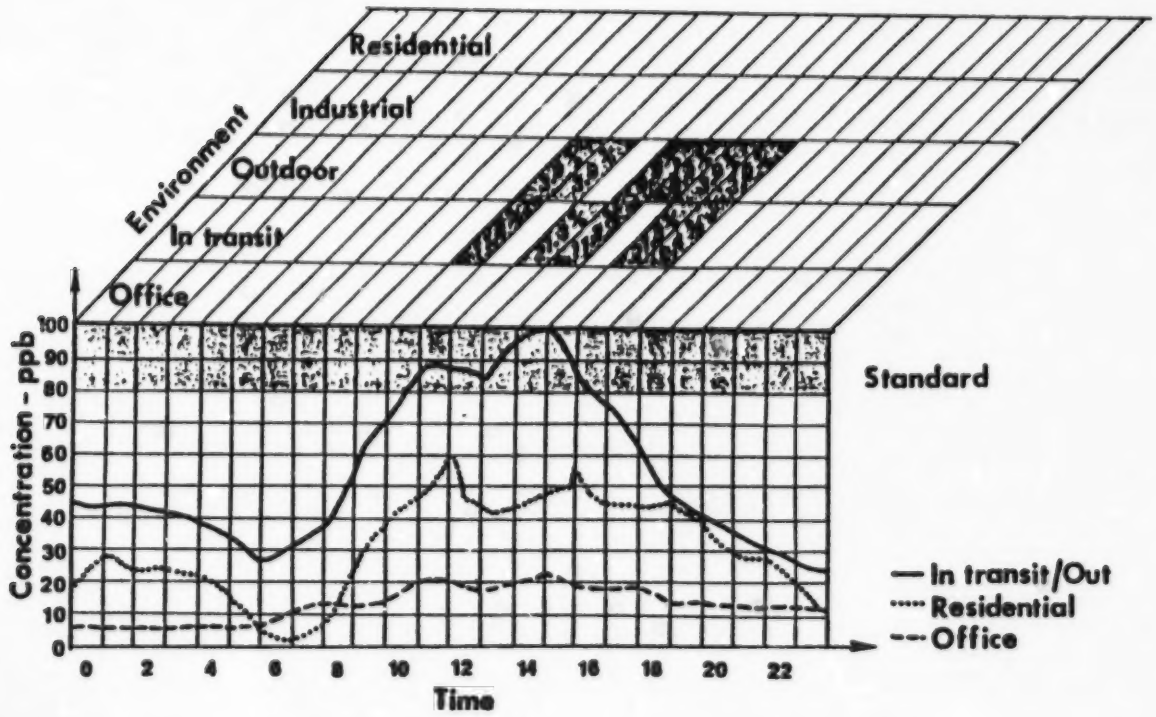
Conceptual view of a three-dimensional space for the estimation of population exposure to air pollutants.



Source: Moschandreas and Morse (1979)

Figure 5

Typical O_3 diurnal variation in four environmental modes illustrating the proportion of population exposed to O_3 concentrations that exceed the 80 ppb standard.



Source: Moschandreas and Morse (1979)

2.2.4 Summary

1) The major determinants of indoor air quality are as follows:
   i) outdoor pollutant concentrations
   ii) indoor pollutant sources and strengths
   iii) air exchange rates of structures
   iv) transfer rates of pollutants to sinks

2) Air exchange rates of buildings are dependent primarily on:
   i) type of ventilation system;
   ii) season;
   iii) structural design (exchange rates may be reduced more than 2/3 in energy-efficient buildings);
   iv) activity and operation patterns

3) Concentrations of nonreactive gases such as carbon monoxide and nitrogen oxide do not vary as much indoors as outdoors when no major indoor sources are present, but the overall average indoor concentration is equal to the average outdoor concentration.

4) Concentrations of reactive gases such as ozone and nitrogen dioxide and particulates do not vary as much indoors as outdoors in the absence of major indoor sources, and the average indoor concentrations are less than outdoors. The absolute difference is dependent on outdoor concentration, building air exchange rate, and the decay or settling rate of the pollutant.

5) Indoor concentrations of some of the air pollutants under consideration in this study, in particular NO_2 and particulates, may be determined predominantly by indoor sources (i.e., unvented gas stoves, smoking, fireplaces, etc.), and indoor concentrations may exceed outdoor concentrations by factors of 2 or more.

6) The GEOMET model (Moschandreas and Stark 1978) appears most suitable of those reviewed for predicting indoor air quality provided adequate time is available to calibrate the model for Canadian conditions and data are compiled to characterize outdoor pollutant concentrations, the housing stock, and indoor pollutant sources.

7) Empirical models of the form described by Pengelly et al (1983) are adequate to describe average conditions for time intervals of 2 to 3 days.

8) The primary location of exposure to air pollutants is indoors. Most Europeans, Americans, and likely most Canadians, spend over 90% of their time indoors.

9) The accuracy of estimates of personal exposure to air pollutants is dependent on individual activity patterns and variations in pollutant concentrations between environments.

10) Models for predicting personal exposure consist of two types:
    i) representative individual models; and
    ii) representative environment models.
    The former are preferable in terms of estimating cumulative exposures, while the latter require input data that is generally more accessible.

11) Numerical models have not yet been applied extensively to characterize population exposure on an individual basis due to data and economic limitations.

2.3 The Economics of Health Impacts: Methodologies and Estimates

The objectives of this section are:

1. To review the methodologies for the economic evaluation of health impacts.

2. To assemble estimates of economic evaluations of health impacts.

3. To discuss the application of the methodologies and estimates to health impacts related to automobile emissions of HC, CO, and NO_x.

Each of these objectives is addressed in the following sections:

2.3.1 Methodologies for Evaluating Health Impacts

In economic approaches to evaluating health impacts no distinction is normally made between health and safety. Health impairment (i.e., illness) and accidents involving physical injury both entail losses to individuals and society. It is the value of these actual or potential losses, without necessarily specifying what causes them, that provide the focus of attention for the economist. This lack of distinction between health and safety is maintained in this report except where otherwise stated, and the term health impacts may be taken to include morbidity and mortality from ill health and injury.

The economic evaluation of health impacts has been approached in two main ways. These can be referred to as:

- output accounting: the value of the output lost and resources foregone because of death, illness, or injury

- welfare accounting: the value of resources individuals would forego (accept) to reduce (increase) a risk of death, illness, or injury if they had the opportunity.

2.3.1.1 Output Accounting

This method is more commonly known as the "human capital" method. Output accounting is a term introduced by the U.S. Institute of Medicine (1981) to emphasize its basis in the measurement of lost output and its similarities to national income accounting. Since "output accounting" is easier to understand, it is the term that is used in this report.

The medical care that people receive when they become ill utilizes a portion of society's resources. These resources, which include labour, buildings, equipment, materials and energy, have alternative uses. They can be used to produce other goods and services that are of value to people. In output accounting, it is normally assumed that the dollar cost of health care services is a reasonably good estimate of the market value of these other goods and services.

In addition to the costs of medical care, output accounting includes the losses attributable to a person's temporary or permanent displacement from the workforce. Again, the market value of the services of labour that are lost are taken to be a satisfactory estimate of the value of the output that they would have generated.

There are two variants of output accounting that should be noted. The first goes beyond the measurement of market-related impacts and includes the valuation of interruptions to such nonmarket activities as housework and leisure. This gives a more complete evaluation of the output foregone through illness, injury, and death.

The second variant, which applies primarily when premature death is involved, subtracts the reduction in consumption to arrive at the net impact on output.

In most cases, losses in output extend over time. The valuation of these losses in a common unit raises the difficult question of discounting, i.e., weighting the losses according to when they are expected such that losses are given less weight the further into the future they occur. While there is agreement among economists about the general validity of this process, there is no consensus on the appropriate discount rate (i.e., set of weights) to

use. This means that the practice of output accounting for valuing health impacts rests, in part, on the resolution of an even more contentious and problematic issue. Typically, sensitivity analysis is employed, so that the effect of changes in the assumed discount rate on the estimate of output losses is made explicit.

The study by the Institute of Medicine (1981) identified the following categories of costs that should be included in an application of output accounting:

- Direct costs -
  expenditures on health services; additional costs for transportation; additional household and relocation expenses; family counselling costs.

- Indirect costs -
  present value of lost earnings from morbidity and mortality; value of time spent visiting physicians and other health professionals by patients, family, and friends.

- Productivity costs -
  reduced productivity on the job, increased absenteeism, unwanted job changes and loss of opportunities for promotion and education. All of these can raise cost per unit output.

- Nonmarket activity costs -
  losses in time from and decline in quality of housekeeping, voluntary activities, and leisure.

The Institute of Medicine Study also included costs attributable to pain and suffering but since these are not output related they have been omitted here. The possibility that some of the above costs could be negative should be noted. This will happen, for example, when premature death reduces the future use of health care services.

2.3.1.2 Welfare Accounting

Welfare accounting is based in the tradition of welfare economics which underlies the theory, if not always the practice, of benefit-cost analysis. In this context, welfare means well-being. Welfare economics deals with "economic" factors that contribute to people's well-being, as they define it. Except incidentally, it has nothing to do with social welfare programs.

A fundamental premise of welfare economics is that people are able to compare anything with anything else in terms of the contribution made to their own welfare: apples with oranges, oranges with shoes, shoes with houses, houses with medical services, medical services with ice cream; everything can be compared with everything else. In a society such as Canada, in which the market system is used extensively to determine what is produced, by whom and for whom, an enormous range of goods and services are available at widely differing prices. These prices are expressed in dollars. The ratio of these prices shows the relative values of the commodities to people. For example, if oranges are 50 cents each and apples are 25 cents each, then at the margin, people value oranges twice as much as apples. If they did not, then they would adjust their purchases until the relative prices did accurately reflect relative values.

The point of this discussion is to show that the valuation of goods and services in terms of dollars is just a convenient way of valuing them in terms of one another. Taken one step further, the valuation in dollars of an improvement or deterioration in health is a valuation in terms of all other goods and services. From the standpoint of welfare economics, this makes sense because everyone is presumed to be able to make comparisons across an unlimited range of alternatives.

The specific comparisons that are considered when welfare accounting is applied to health impacts are between changes in the <u>risk</u> of mortality and/or morbidity and the goods and services available to people. Four possibilities arise. The change in risk can be positive or negative, and the goods and services (represented by dollars) can be given up or received. People's value of changes in health risks can therefore be considered as:

1. Their willingness to pay for a reduction in risk.

2. Their willingness to accept compensation to forego a reduction in risk.

3. Their willingness to accept compensation for an increase in risk.

4. Their willingness to pay to avoid an increase in risk.

As explained in Appendix 3, it is possible for each of these four measures to be different for the same change in risk. This will generally be the case when the change in risk is exogenous to the individual and there are no other opportunities for the individual to exchange dollars, either through changes in consumer expenditures or working conditions, for further changes in risk. When these opportunities do exist, say through the purchase of smoke detectors or a change in employment, it can be shown that measures 1 and 2 are the same and measures 3 and 4 are the same. However, this still leaves two measures for valuing a change in risk and this should be acknowledged in applications of the welfare accounting methodology. That this is seldom done is apparent from the more common use of the term "willingness-to-pay" to refer to this approach, even though, under some circumstances, the more appropriate valuation concept might be willingness-to-accept compensation.

(It should be noted that the difference between the willingness-to-pay and willingness-to-accept measures of value is "very small" when the change in risk being valued is also "very small". This theoretical result, which has been called into question by some recent empirical studies, may provide a justification for neglecting the differences in some applications).

Implementation of welfare accounting for valuing health impacts has been attempted by two methods. One is the imputation of health risk values from observed market behaviour where risk is believed to be an important factor. The other is through surveys designed to elicit people's valuations through responses to hypothetical questions. Examples of these methods are reviewed in Section 2.3.2.

2.3.1.3 A Comparison of Output Accounting and Welfare Accounting

This comparison is based entirely on a consideration of the conceptual issues. Ease of implementation, which is of considerable concern to the wider study of which this report is a part, will be addressed in the subsequent review of studies. The comparison proceeds according to the following:

1. ease of comprehension
2. consistency with benefit-cost analysis principles
3. availability of information
4. ex ante versus ex post
5. potential range of impacts covered
6. distributional considerations
7. discounting
8. potential role in policy analysis

Ease of Comprehension

Except those trained in welfare economics, most people are likely to find output accounting easier to understand. This is important given the role that estimates of the value of health impacts are intended to play in policy and program decisions. Output accounting yields estimates that everyone recognizes as partial. No one is likely to value human life solely in terms of output and everyone realizes there are other important dimensions to be considered.

The broader scope of welfare accounting, at least in principle, allows many if not all of these other dimensions to be incorporated in the evaluation of health impacts. However, this may not be appreciated by noneconomists and can lead to misunderstanding and confusion.

Consistency with Benefit-Cost Analysis Principles

During the past twenty years or so benefit-cost analysis (BCA) has been used increasingly by government departments to assess proposed programs and projects. The Treasury Board of Canada has issued guidelines on BCA explicitly for this purpose (Treasury Board 1976, 1979). A major advantage,

therefore, of welfare accounting is its consistency with the principles of BCA. This has been most forcefully argued by Mishan (1971, 1982) and is a position widely accepted by economists.

Availability of Information

Output accounting requires only that those estimating the value of health impacts be well-informed about risks and consequences. In contrast, welfare accounting values these risks and consequences from the standpoint of those affected. If these people are poorly informed, then it is highly questionable as to what significance, if any, should be given to valuations that reflect a serious lack of information. Mishan notes that in BCA it is customary to rely on individuals' own evaluations of effects on their welfare, whatever their level of information. (Mishan 1971). However, this seems an inadequate justification for continuing the practice.

Ex ante versus Ex post

In output accounting, health impacts are valued on the assumption that they have occurred or will occur. Risk is not a factor. The approach identifies and measures the output foregone and resources utilized in the event of illness, injury, or death. It is in this sense that the valuation is ex post or after the fact.

In contrast, welfare accounting values changes in the risk of illness, injury, or death. As such it is ex ante, i.e., before the fact. Broome (1978) has argued that, for any individual, the ex ante and ex post evaluations of health impacts may be substantially different. He interprets the ex post evaluation as, in some sense, the correct evaluation and argues that ex ante evaluations are only useful as estimates of these. In the case of death, when willingness-to-accept compensation is considered, the ex post value is infinite. Therefore Broome rejects all ex ante welfare accounting evaluations when death is involved.

The validity of Broome's argument rests on his claim that ex ante evaluations are merely estimates of the true ex post values. This makes sense in relation to benefits arising, for example, from travel when a new road is

being contemplated. Evaluation of the benefits prior to construction will be based on estimated travel patterns with and without the new road. After the decision to build or not to build is taken, it becomes possible to measure ex post one of the actual travel patterns (i.e., with or without the road) and compare this with the ex ante estimate. In this case it is clear that the accuracy of the ex ante estimate of travel is only as good as its approximation to the true ex post value.

Broome has extended this argument to decisions in which mortality risks play an important role. However, in doing so he concentrates not on the change in risk as the variable to be estimated, but on the value of the loss of life itself. Otherwise the correct parallel would be between ex ante and ex post estimates of changes in risk and not, as he suggests, the ex ante and ex post estimates of the value of life.

It is precisely this, i.e., the valuation of risk rather than the valuation only of the consequences of incurring a risk, that distinguishes welfare accounting from output accounting. As such it seems quite appropriate to estimate individuals' evaluation of the risks and to use the estimates in project or program assessment.

Potential Range of Impacts Covered

There is a major difference in the potential ranges of health impacts that the two methodologies can encompass. By definition, output accounting is restricted to impacts that give rise to changes in the output of goods and services. The pain and suffering of those who experience the health impacts and the grief borne by family and friends are excluded except to the extent they are mitigated through the consumption of medicines, and the use of counselling services, and possibly through their effects on productivity. Also, people not in the workforce such as children and the aged can be ill without there being any direct loss of output.

In welfare accounting, it is changes in the risk of health impacts and all their possible consequences that one seeks to value. These include consequences to third parties as well as to the individuals at risk. If many of these consequences are not output related then the difference in the range

of coverage between the two methodologies could be considerable.

Distributional Considerations

The evaluation of health impacts by output or welfare accounting is always based on some assumed distribution of wealth and income. This is normally that of the status quo.

Lost output due to time off from work is valued, in output accounting, at the wage rate of the worker. Obviously, this will vary according to the income level of the worker. Similarly, the prices of the medical services consumed will reflect the prevailing distribution of incomes and wealth. The result is that the value of output lost due to illness, injury, or death will be based on a specific distribution of income. A different distribution might yield a very different evaluation of health impacts.

Welfare accounting relies on payment or compensation measures for valuing changes in the risk of health impacts. In economic terms, willingess-to-pay is always constrained by ability to pay. (The fact that willingness-to-accept compensation bears no such constraint partly explains the differences in these two measures). But even the compensation people require to willingly accept the risk of adverse health impacts is likely to be related to their income. Consequently, the welfare accounting measures of the value of health impacts are based on a given distribution of income and wealth. If this were different, the valuation would be too.

Discounting

Estimates of valuations that extend over time can best be compared by conversion to an equivalent capital or present value sum. This is accomplished by discounting the values. In output accounting the rate of discount should reflect the rate of return that the resources, if released from health care or made available through greater output, would earn when allocated throughout the economy. It is the capacity of the economy to transform current output into an expanded future output that should determine the discount rate used in output accounting.

In welfare accounting, the comparison of present and future losses from adverse health impacts is implicitly performed by each individual when assessing the value they place on increased risk. Insofar as it is the value of the risk of these impacts that is the proper subject of measurement, then the value of the possible future losses will be discounted by those subjected to the risks. No further discounting is necessary or appropriate since the value of the increased risk is already expressed as a capitalized, present value.

Potential Role in Policy Analysis

The primary interest of those who have contributed to the development of output and welfare accounting has been in the generation of value estimates that are relevant to public sector decision making. This applies as much to the work of theoreticians as it does to that of practitioners.

It should be clear from previous sections of this report, that the two methodologies yield estimates of two quite different, though related, magnitudes. (The nature of this relationship is discussed in the next section). The question arises, therefore, as to the role of each of these magnitudes in policy analysis.

Economists would generally argue that, since welfare accounting is consistent with BCA, and BCA is now well-entrenched in public sector decision making, a strong case can be made for selecting this methodology over output accounting. "It is the only economically justifiable concept" (Mishan 1971). Practical difficulties of implementing the methodology may require a pragmatic withdrawal from this position, though again Mishan warns that "there is more to be said for rough estimates of the precise concept than precise estimates of economically irrelevant concepts". (Mishan 1971).

The thrust of Mishan's argument is not that output accounting estimates are economically meaningless, only that they are inconsistent with the theoretical foundations of BCA. It is quite conceivable that others involved in policy analysis might attach considerable importance to estimates of foregone output from health impacts. Economists can point out that such estimates cannot, without qualification, be used in a BCA or compared with

the results of a BCA. They are derived from a different perspective and a different set of principles. However, economists cannot insist that their formulation of the policy problem, and hence their view on what information is appropriate for policy analysis, is the only legitimate one. This is a question worthy of further consideration.

The Relationship Between Welfare Accounting and Output Accounting

Several authors have considered the relationship between welfare accounting and output accounting. (Landefeld and Seskin 1982, Linnerooth 1979, Conley 1976). This has usually been approached through comparisons between a person's willingness-to-pay to reduce the risk of mortality and their expected lifetime earnings. Linnerooth shows that no general conclusions can be reached regarding this relationship and that it all depends on the nature of the individual's utility function. (A utility function is a mathematical statement of the relationship between an individual's welfare and all that it depends upon).

However, under certain restrictive assumptions it can be shown that the welfare-based total value that a population would be willing to pay to reduce the risk of mortality equals the present value of the expected loss of incomes. A critical assumption for this result to obtain is that people seek only to maximize the expected value of discounted lifetime income. They will therefore be willing to pay as much to reduce the risk of losing all their income as the expected value of their income loss in the event of their death. (Landefeld and Seskin 1982). It is because people have other objectives in life besides income maximization and that a person's illness, injury, or death matters to others, that welfare accounting and output accounting can yield very different values for human health impacts.

The Adjusted Willingness to Pay/Human Capital Approach

Faced with the methodological superiority of welfare accounting and the greater ease of implementation of output accounting, Landefeld and Seskin (1982) have developed a third option for valuing risks to human life. They refer to it as an "adjusted willingness to pay/human capital approach". Such an approach embodies characteristics of both welfare accounting (WTP) and

output accounting (HK). It represents an attempt to derive "clear, consistent and objective values for use in cost-benefit analysis of policies affecting risks to life". (Landefeld and Seskin).

Landefeld and Seskin propose the following equation for valuing lives:

$$VL = \left[\sum_{t}^{T} \frac{Y_t}{(1+r)^t}\right] a \qquad 1.$$

where: VL = the value of an individual life

T = remaining lifetime

Y_t = after-tax income

$= L_t + NL_t$

where L_t = labour income (this may include the imputed value of housekeeping services)

NL_t = nonlabor income

r = individual's opportunity cost of investing in risk-reducing activities

a = risk aversion factor

In discussing equation 1, Landefeld and Seskin write:

> "As with the standard HK approach, it includes only economic losses associated with death; hence, the resulting estimates can be considered a lower bound for the value per statistical life. As with the revealed-preference WTP method, it is based on WTP for small changes in risk of death from the perspective of the individual rather than from that of society. ..... Future benefits are given by the summation over t of an individual's after-tax income (Y_t). Y_t includes both labour income (L_t) and non-labor income (NL_t), but does not include measures of nonmarket activities or leisure. The discount rate (r) used to calculate the present value of future income represents the individual's (as opposed to society's) opportunity cost of investing in risk-reducing activities using the household's assets. Since, again, it is the individual's perspectivie that is important, the relevant rate of return is after taxes. A risk-aversion factor (a) is also applied for the same reason that it is thought to be relevant in the revealed-preference WTP estimates, namely, that persons should be at least as risk-averse with regard to loss of life as they are with regard to other assets in their investment portfolios".

Landefeld and Seskin apply their approach to data for the U.S. and demonstrate that it is indeed comparatively simple to implement. It can serve as a lower bound to a welfare accounting estimate of risk to human life. Furthermore, the same approach can be applied to risks of morbidity, although it would be necessary to add in medical expenses which Landefeld and Seskin were able to omit when only considering mortality.

From a methodological point of view, there are some questions to be asked of the Landefeld and Seskin approach. They only include income net of tax on the grounds that this is the value of foregone income to the individual. Even if one assumes that a person's benefit from government services is independent of taxes paid, other people must be presumed to lose if there is a decline in income taxes. Presumably, they would be willing to pay up to this amount to prevent the loss in tax revenues. Consequently, a complete estimate of individuals' willingness to pay to avoid a loss of employment from morbidity or mortality should include the unpaid income taxes as well as the labour income.

Similarly, nonlabour income should not be included since, when a person dies, this income accrues to the beneficiaries of the estate. The willingness to pay of someone to avoid a loss in nonlabour income is therefore matched by the willingness to pay of others to receive an equivalent gain. These sums cancel out and should therefore be excluded.

In conclusion, Landefeld and Seskin's compromise approach is a reasonable way of estimating the lower bound of an individual's willingness to pay to avoid death. However, by excluding income taxes and including nonlabour income it neglects entirely the willingness to pay of others for the avoidance of a person's death. This omission is inconsistent with the tenets of benefit-cost analysis which demand that benefits to all individuals within the relevant jurisdiction be considered.

2.3.2 A Survey of Estimates

In a review of the cost of illness, Hu and Sandifer reported over 200 studies that utilize the output accounting methodology. (Hu and Sandifer 1981). Another annotated review by Drummond (1981) describes 101 studies, many of which employed some form of output accounting. The number of studies using welfare accounting is considerably less, in the order of 20 or so.

Very little empirical work, with either methodology, has been done in Canada. Colin Lay, of the health administration department of the University of Ottawa, is engaged in designing standard costing practices for hospitals (Hayes and Lay 1983) and is currently analysing cost data provided by the Ottawa Civic Hospital. He is in the process of allocating the hospital's costs to departments, which will provide a reasonable basis for estimating in-patient costs by disease for that hospital. Kingston General Hospital and University Hospital in London are both adopting standarized practices for costing which will allow the hospitals' costs to be related to specific diseases.

Another approach to estimating disease specific hospital costs is that of Morris Barer. Using data from hospitals in Ontario, he has developed econometric estimates of hospital costs for 237 diagnosis specific categories (Barer 1981). A somewhat similar, though less sophisticated, approach was taken by Fraser, Spasoff and Prime (1976) who estimated the hospital and nonhospital medical costs by diagnostic category for Ontario in 1971.

Only one Canadian study using the welfare accounting methodology has been identified. This is an unpublished paper by Michael Hinton (1980) written when he was a graduate student at the University of Toronto. His analysis of Canadian labour market data follows in the tradition established by Thaler and Rosen (1976) and others of estimating the extent to which differences in job risks are reflected in wage differentials.

The comparative lack of welfare accounting estimates of the value of health impacts stands in sharp contrast to the number of reviews of the studies that have been published. (Bailey 1980, Gunderson and Swinton 1981, Butcher 1981, Blomquist 1982, Landefeld and Seskin 1982, and Violette and Chestnut 1983).

Other studies, directed at somewhat broader issues, have also included literature reviews. (Fisher, Hamilton and Scotchmer 1979, Hamilton 1979, Viscusi 1979, Fisher 1981). The review by Violette and Chestnut in 1983 is especially comprehensive. It succeeds in meeting its authors' goal which is "to provide enough detail on how each study was conducted to allow the reader to have a true feel for the different willingness-to-pay estimates and their applicability to different policy questions". (Violette and Chestnut 1983).

In light of this plethora of reviews of a rather sparse welfare accounting literature, and the less problematic nature of output accounting, the strategy adopted in this report is as follows:

1. To give a reasonably detailed account of the seminal empirical studies in output accounting and welfare accounting of health impacts (Cooper and Rice 1976, Thaler and Rosen 1976 respectively).

2. To note the significant points at which the other welfare accounting studies differ from Thaler and Rosen.

3. To pay special attention to the studies based on Canadian data.

4. To summarize the estimates of welfare accounting measures of the value of health impacts. (No such summary is offered for the output accounting estimates. This is due to the large number of studies involved and the highly specific nature of many of them making summarization inappropriate).

2.3.2.1 Output Accounting: The Economic Cost of Illness

In 1966, Dorothy Rice published her much referenced paper, "Estimating the Cost of Illness", (Rice 1966). Ten years later she coauthored a paper with Barbara Cooper (Cooper and Rice 1976) in which new estimates of the costs of illness, in the USA, were reported. The paper gives a clear exposition of the methodology used. It is this latter paper which is considered here.

Table 2.8 presents Cooper and Rice's estimate of the total economic costs of illness in the USA by diagnosis, for the year 1972. The three categories of cost components, i.e., direct costs, morbidity costs, and mortality costs,

Table 2.8

Total Economic Costs: Estimated Direct Costs, Indirect Cost of Morbidity and Mortality, with Present Value of Lifetime Earnings Discounted at 4 percent and 6 percent, by diagnosis, 1972
U.S. Dollars

Diagnosis	Amount (in millions)				Percentage distribution			
	Total	Direct Costs	Indirect Costs		Total	Direct Costs	Indirect Costs	
			Morbidity	Mortality			Morbidity	Mortality
	4 percent							
Total	$188,789	$75,231	$42,323	$71,235	100.0	100.0	100.0	100.0
Infective and parasitic diseases	3,443	1,412	1,200	831	1.8	1.9	2.8	1.2
Neoplasms	17,367	3,872	862	12,633	9.2	5.1	2.0	17.7
Endocrine, nutritional, and metabolic diseases	5,930	3,436	1,137	1,357	3.1	4.6	2.7	1.9
Diseases of the blood and blood-forming organs	921	491	220	210	.5	.7	.5	.3
Mental disorders	13,917	6,985	6,179	753	7.4	9.3	14.6	1.1
Diseases of the nervous system and sense organs	10,951	5,947	3,944	1,060	5.8	7.9	9.3	1.5
Diseases of the circulatory system	40,060	10,919	6,417	22,724	21.2	14.5	15.2	31.9
Diseases of the respiratory system	16,454	5,931	7,089	3,434	8.7	7.9	16.7	4.8
Diseases of the digestive system	17,487	11,100	2,606	3,781	9.3	14.8	6.2	5.3
Diseases of the genitourinary system	6,456	4,471	1,249	736	3.4	5.9	3.0	1.0
Complications of pregnancy, childbirth, and the puerperium	2,932	2,607	245	80	1.6	3.5	.6	.1
Diseases of the skin and subcutaneous tissue	2,052	1,525	460	67	1.1	2.0	1.1	.1
Diseases of the musculoskeletal system and connective tissue	8,948	3,636	5,103	209	4.7	4.8	12.1	.3
Congential anomalies	1,903	381	238	1,284	1.0	.5	.6	1.8
Accidents, poisonings, and violence	26,678	5,121	3,883	17,674	14.1	6.8	9.2	24.8
Other	13,294	7,398	1,494	4,402	7.0	9.8	3.5	6.2

Table 2.8 (continued)

Diagnosis	Amount (in millions)				Percentage distribution			
	Total	Direct Costs	Indirect Costs		Total	Direct Costs	Indirect Costs	
			Morbidity	Mortality			Morbidity	Mortality
	6 percent							
Total	$174,934	$75,231	$42,323	$57,380	100.0	100.0	100.0	100.0
Infective and parasitic diseases	3,234	1,412	1,200	622	1.8	1.9	2.8	1.1
Neoplasms	15,641	3,872	862	10,907	8.9	5.1	2.0	19.0
Endocrine, nutritional, and metabolic diseases	5,717	3,436	1,137	1,144	3.3	4.6	2.7	2.0
Diseases of the blood and blood-forming organs	875	491	220	164	.5	.7	.5	.3
Mental disorders	13,782	6,985	6,179	618	7.9	9.3	14.6	1.1
Diseases of the nervous system and sense organs	10,703	5,947	3,944	812	6.1	7.9	9.3	1.4
Diseases of the circulatory system	37,430	10,919	6,417	20,094	21.4	14.5	15.2	35.0
Diseases of the respiratory system	15,764	5,931	7,089	2,744	9.0	7.9	16.7	4.8
Diseases of the digestive system	16,931	11,100	2,606	3,225	9.7	14.8	6.2	5.6
Diseases of the genitourinary system	6,344	4,471	1,249	624	3.6	5.9	3.0	1.1
Complications of pregnancy, childbirth, and the puerperium	2,914	2,607	245	62	1.7	3.5	.6	.1
Diseases of the skin and subcutaneous tissue	2,040	1,525	460	55	1.2	2.0	1.1	.1
Diseases of the musculoskeletal system and connective tissue	8,913	3,636	5,103	174	5.1	4.8	12.1	.3
Congential anomalies	1,375	381	238	756	.8	.5	.6	1.3
Accidents, poisonings, and violence	21,649	5,121	3,883	12,645	12.4	6.8	9.2	22.0
Other	11,625	7,398	1,494	2,733	6.6	9.8	3.5	4.8

Source: Cooper and Rice (1976)

were defined and estimated by Rice and Cooper as follows:

Direct costs: expenditures for prevention, detection, treatment, rehabilitation, research, training, and capital investment in medical facilities. Information on direct costs was obtained from estimates published annually by the Social Security Administration. The estimates include costs for hospital care, physicians' services, drugs and drug sundries, eyeglasses and appliances, and nursing home care. For each type of expenditure, the total expenditure was distributed, by diagnosis, on the basis of utilization and cost data, using the same data sources for each diagnosis.

Morbidity costs: losses from morbidity are incurred when illness results in absence from employment, prevents a housewife from performing her duties, or results in disability that prevents someone from working at all. The lost earnings and the dollar value of the unperformed housekeeping services are the morbidity costs.

To measure these costs, Cooper and Rice assumed that people too ill to work would not necessarily have been fully employed had they not been ill. Instead they assumed that their employment would have corresponded to that of the general population, which they analysed by age group and sex. Cooper and Rice also assumed that, if these people had been in the labour force, there would have been no impact on employment rates or earning levels.

For the valuation of housewives' services, the results of a time and motion study were combined with the relevant market wages for various housekeeping services. This gave estimates of the cost of replacing the housewife's efforts with persons employed at market wages to do the same work.

Allocation of morbidity costs by diagnosis was problematic.

Cooper and Rice relied on patients for diagnostic information, the accuracy of which is questionable. Multiple diseases required the use of arbitrary cost allocation procedures.

Mortality costs: The present value of future lost income and the present value of lost housekeeping services were used as measures of the foregone output from premature death.

Mortality costs were calculated by multiplying the number of deaths (by age, sex, and race) by the present values of lifetime earnings and housewives' services. The method takes account of the life expectancy for different age, sex, and race groups, varying labour force participation rates, the current changing pattern of housewives' services, and the discount rate.

Discussion

Commenting on their own work, Cooper and Rice observe the following:

i) The costs of pain and suffering, a major category of costs, are not included on the grounds that a suitable methodology is not available.

ii) In valuing housewives' services, the psychic value of a housewife to her family or society was not considered.

iii) Their methodology for valuing mortality costs has been objected to because men are valued higher than women, whites higher than other races, and those in the employed ages higher than the very young and very old.

There is an implication in Cooper and Rice's paper that these comments reflect deficiencies in their work. This seems not to be the case, providing one is seeking only an estimate of output lost and resources foregone from illness, injury, and death. In other words, it is essential to recognize what output accounting can provide. It can measure the costs of ill health but only in this restricted sense of lost output and foregone resources.

Since people do not seek good health merely to increase economic output, output accounting measures bear no necessary relation, except possibly as a lower bound, to what they are prepared to pay to ensure good health. If society can prevent the losses from ill health that output accounting reveals, by reallocating its resources such that overall output is increased, then that is probably worthwhile. (Note, to ascertain this, marginal costs by diagnosis rather than the average costs estimated by Cooper and Rice, are required). However, a net increase in output is neither a necessary or a sufficient condition for the economic justification of health care programs. This is the message of welfare accounting and it should not be forgotten.

Hence, in Cooper and Rice's work, the omission of pain and suffering costs is only a problem to the extent that a source of lost output (e.g., through reduced productivity) may have been overlooked. Similarly, the psychic value of a housewife's services would seem to have no place in the output accounting framework. And, the widely different economic productivity of various groups in society is a fact of life. Arguably, it should have no normative significance for determining public programs concerned with health. However, estimates of lost output from ill health should reflect this circumstance. (The implications for productivity from a more egalitarian social structure deserve to be studied in their own right.)

In conclusion, Cooper and Rice, and many others besides, have shown that reasonably consistent and reliable estimates of the economic costs of ill health can be obtained using the output accounting methodology. What is lacking is a coherent statement of the policy relevance of such estimates.

2.3.2.2 Canadian Estimates of Diagnosis-Specific Health Care Costs

Barer (1981) formulated and estimated two models of hospital, inpatient costs. The models differ only in that one relates costs to cases (separations) per year and the other relates costs to inpatient days.

Barer's objective was to "explain" variation in hospital costs by an analysis of variables such as case flow rate; occupancy rate; noninpatient care activities such as education, research, and outpatient functions; and the age, sex, and diagnostic mix of the patients treated. The use of multiple

regression analysis allowed him to estimate the relative importance of each variable in accounting for the variation in costs among hospitals.

With the results obtained, Barer was able to estimate the diagnosis-specific marginal hospital costs. His procedure involved changes in the values of specific variables related to the treatment of each disease and the calculation of the implied changes in costs per patient day or costs per case.

Equations 2 and 3 were used by Barer to specify his models:

$$TC = p_1B + p_2B^2 + p_3C \qquad 2.$$

where: TC is total inpatient cost
B is rated bed capacity
C is cases per year

$$TC = p_4B + p_5B^2 + p_6D \qquad 3.$$

where: D is total patient days per year

Barer assumed the coefficients p_1, p_2, p_4 and p_5 to be constants, but p_3 and p_6 to depend on several variables relating to hospital activities and patient mix. He estimated their values with Equations 4 and 5.

$$p_3 = a_0 + a_1CMPXCI + a_2SPCLCI + a_3EDRAT + a_4DEPRAT + a_5F_1 +$$
$$\ldots + a_{12}F_8 + a_{13}OUTXPR + a_{14}WAGE + a_{15}SERV + a_{16}ALS \qquad 4.$$

$$p_6 = b_0 + b_1CMPXCI + b_2SPCLCI + b_3EDRAT + b_4DEPRAT + b_5F_1 +$$
$$\ldots + b_{12}F_8 + b_{13}OUTXPR + b_{14}WAGE + b_{15}SERV + b_{16}ALS \qquad 5.$$

where: the variables are briefly defined in Table 2.9

Substituting Equations 4 and 5 respectively into Equations 2 and 3, and transforming the latter two equations into their respective per unit specifications (i.e., average cost per separation for Equation 2, and average

Table 2.9

Variable Acronyms and Brief Descriptions

Dependent variables	
CASEX	inpatient cost per hospital separation
CASEXD	inpatient cost per hospital separation in 1969 dollars
DAYEX	inpatient cost per hospital day
DAYEXD	inpatient cost per hospital day in 1969 dollars
Independent variables	
EDRAT	educational activity expenditure as share of total hospital operating costs
DEPRAT	non-departmental (e.g., interest and depreciation) expenditure as share of total hospital operating costs
OUTXPR	expenditures of organized outpatient and emergency departments, and other ambulatory care costs, as share of total hospital operating costs
$F_1 \ldots F_8$	factor scores from a factor analysis of the inpatient age-sex distribution of separations across hospitals. See Appendix A for relevant loadings on original age-sex categories.
CMPXCI	complexity of hospital separations mix
CMPADJ	CMPXCI adjusted for aggregate provincial changes in case mix (by using base year case complexities)
SPCLCI	a measure of hospital specialization; an indication of the degree to which a hospital is limited in its capacity to handle a wide range of case types
SERV	an indicator of the relative cost of a hospital's personnel (service) mix.
WAGE	an indicator of a hospital's relative wage level
ALS	average length of stay
OCC	occupancy rate
B	rated bed capacity

Source: Barer (1981)

cost per patient day for Equation 3) gave the following:

CASEX = TC/C

where: CASEX is average inpatient cost per separation
TC is total inpatient cost
C is the number of inpatient discharges and deaths

$$CASEX = p_1 B/C + p_2 B^2/C + a_0 + a_1 CMPXC + a_2 SPCLCI + a_3 EDRAT + a_4 DEPRAT + a_5 F_1 + \ldots + a_{12} F_8 + a_{13} OUTXPR + a_{14} WAGE + a_{15} SERV + a_{16} ALS \quad 6.$$

DAYFEX = average inpatient cost per day = TC/D

where: DAYFEX is average inpatient cost per day
D is total inpatient days

$$DAYFEX = p_4 B/D + p_5 B^2/D + b_0 + b_1 CMPXCI + b_2 SPCLCI + b_3 EDRAT + b_4 DEPRAT + b_5 F_1 + \ldots + b_{12} F_8 + b_{13} OUTXPR + b_{14} WAGE + b_{15} SERV + b_{16} ALS \quad 7.$$

Equations 6 and 7 represent Barer's belief about how hospital costs behave. He estimated these equations using data from 182 Ontario hospitals for each of the 6 years, 1969 - 1974. Subsequently, he pooled the data and reestimated the equations.

Using his results Barer proceeded to estimate the diagnosis-specific marginal inpatient costs as follows. He changed each hospital's case mix by altering its number of separations or patient days in one diagnostic category at a time. The difference between the estimated hospital costs before and after the changed case mix (a 10% decline in separations or patient days) gave the diagnostic-specific marginal costs.

Having obtained marginal-cost estimates for each hospital and each diagnostic category, he calculated the marginal cost over all hospitals and years, by weighting the hospital and year-specific estimates according to case load. Barer's results are given in Appendix 2. Definitions of the diagnostic

categories are also given in Appendix 2.

Barer makes several observations on his results. Firstly, the marginal costs based on concurrent reductions in case load and in bed stock substantially exceed those based only on case load adjustments. Secondly, the low-cost diagnostic categories such as 110, 118, 219 and 229 are the categories that would be expected to yield low costs: hypertrophy of tonsils and adenoids, diseases of teeth and supporting structures, generally uncomplicated external head and neck wounds, and toxic effect of chiefly nonmedical substances. At the other end of the cost scale, high-cost diagnostic categories such as 185: spina bifida and congenital hydrocephalus are both very serious problems. On this basis one can have reasonable confidence in the relative magnitudes of the marginal costs. Their absolute values may be less accurate, although the care with which they were estimated, and the explanatory power of Barer's cost equations, suggest that they can be utilized with some confidence in disease costing studies.

In a study for the Ontario Council of Health, Fraser, Spasoff and Prime (1976) prepared estimates of the direct and indirect costs associated with 18 major diagnostic categories for Ontario in 1971. (These categories are aggregations of the 237 categories used by Barer). They also disaggregated hospital and physician costs according to the 188 categories of the Canadian Hospital Morbidity List.

Fraser, Spasoff and Prime adopted a similar technology of the burden of ill health similar to that of Cooper and Rice (1976):

A. Direct Costs:

1. Personal health care:
   a) Hospital Costs
   b) Nonhospital medical care costs
   c) Other costs

2. Nonpersonal, public health care costs

3. Education

4. Research

B. Indirect Costs:

1. Monetary:
   a) Mortality
   b) Morbidity:
      i) Treated (institutional)
      ii) Untreated

2. Nonmonetary (pain, sorrow, etc.)

Because of the lack of appropriate data, the estimated costs, which are summarized in Appendix 2 do not fully cover all of the items noted above, and the authors make no claim that they do. A brief account of their methodology for each category of costs follows.

Hospital Costs

An average per diem cost by type of institution was multiplied by the number of separated days of care in Ontario's hospitals by diagnostic category. This approach assumes the same cost per inpatient-day for all diagnostic categories and the cost is independent of case severity.

Physicians Services

Estimates were made of the value of services covered by the provincial medical care insurance plan (including private medical care) and other services such as those provided for the Workmen's Compensation Board, the Canadian Forces and prison inmates. The former category accounted for 96% of total physician services and only the methodology employed for estimating these services is described here.

The approach employed a sample of 138 physicians who submitted information on the primary diagnosis associated with each service provided during a total of four study weeks. O.H.I.P. expenditures are coded by the speciality of the physician and not the diagnosis of the patient. The sample data was used to calculate the percentage distribution of O.H.I.P. billings among diagnoses for each speciality. These percentages were applied to the estimated total O.H.I.P. payments for each speciality in 1971. (O.H.I.P. began in 1972 so inflation-adjusted data for this year was used).

Despite several limitations relating primarily to the small sample size, the results do give an estimate of diagnosis specific nonhospital medical service costs.

Drugs and Pharmaceuticals

Fraser, Spasoff and Prime relied on a study of drug costs performed by the Ontario Ministry of Health. The study estimated the gross expenditures on drugs in Ontario during 1971. (It is unclear from Fraser, Spasoff and Prime whether this included all drugs or only prescription drugs. The OMH study itself has not been reviewed). Fraser, Spasoff and Prime adjusted the expenditure data to avoid double counting (e.g., to remove expenditures on drugs in hospitals that were already included in their estimates of hospital costs). They used U.S. information provided by Barbara Cooper on the allocation of drugs by diagnostic category to allocate the expenditures on drugs in Ontario to diagnostic category.

Morbidity and Mortality

Fraser, Spasoff and Prime used an approach similar to that of Cooper and Rice (1976) to estimate the indirect burden of ill health stemming from mortality and morbidity. Taking account of the age and sex of those suffering illness or premature death, they estimated the value of foregone earnings. Their estimates were limited to hospital inpatients because of the lack of data for nonhospitalized patients. A value for the services provided by housewives that were lost because of ill health was also included. For this purpose it was assumed that they could have earned the same income as those women of the same age who were employed in the labour force.

2.3.2.3 **Welfare Accounting - The Value of Saving a Life**[1]

Three principal methods have been used to implement the welfare accounting methodology:

- wage/risk analysis using labour market data
- consumer market studies
- contingent market approaches.

The first two of these methods are based on the theory of hedonic pricing. This theory posits that the prices paid for goods and services depends upon their characteristics. The price of a commodity is equal to the summation of the prices of these individual characteristics. (In this analysis labour is regarded as a commodity.)

Treating risk as a characteristic, it is possible to estimate the price associated with it and to interpret this as peoples' willingness to pay for risk reduction and/or their willingness to accept compensation to incur risk.

1 This section draws extensively from Violette and Chestnut (1983)

In contingent market approaches, questionnnaires are used to elicit people's evaluation (i.e., WTP and WTA) of changes in the risk of illness, injury, and/or death.

The most frequently cited example of wage/risk analysis using labour market data is the study by Thaler and Rosen (1976). This paper is based on the theoretical work of Rosen (1974), illustrated in Figure 6.

Workers are assumed to have different preferences for wages and the probability of injury. In Figure 6 indifference curves for two workers, Y and Z, are shown. (These curves show alternate combinations of wages and the probability of injury among which the workers are indifferent). Correspondingly, firms face different opportunities for varying the probability of injury and the level of wages such that their profits are unaffected. Iso-profit curves for two firms, A and B, are shown in Figure 6.

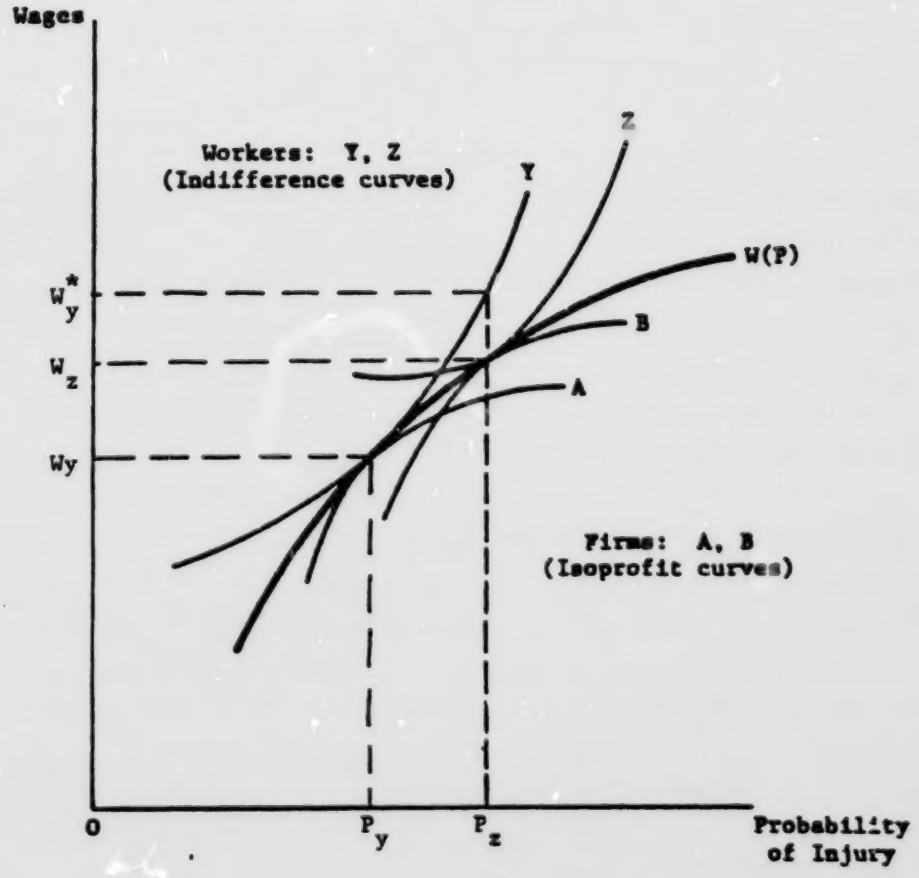
In market equilibrium workers and firms are doing the best for themselves. Each arrives at the trade-off between wages and probability of injury that cannot be improved upon under the circumstances. Worker Y works for firm A and worker Z works for firm B. An analysis of labour market data will reveal the function $W_{(p)}$. This shows the rate at which wages are traded for the probability of injury in the labour market. It does <u>not</u> show the indifference curve for any worker or iso-profit curve for any firm. Consequently, it can only be used to show how workers value very small variations in risk. (Their valuation of larger changes in risk should be based on discrete portions of their indifference curves, and not just on the slopes at specific points on these curves. See Appendix 3 for an example.)

Estimation of $W_{(p)}$ by Thaler and Rosen, and others who have undertaken similar studies, proceeds by specifying an equation of the following type:

Figure 6

Market Equilibrium

Thaler and Rosen (1976)



$$w = a + b.r + c_1x_1 + c_2x_2 .... + c_nx_n \qquad 8.$$

where: w = wage rate
a = constant
r = risk
$x_1 \ldots x_n$ = nonrisk variables that determine the wage rate
b = coefficient for the risk variable
$c_1 \ldots c_n$ = coefficients for the nonrisk variables

Squared variables and/or logarithms are sometimes used to represent nonlinearities in the $W_{(p)}$ function. These do not change the basic approach, and are not considered here. Equation 8 assumes that the wage-risk function $W_{(p)}$ in Figure 6 is linear.

Suppose the value of b is estimated at .0352 for a risk variable r measured in fatalities per 100,000 workers per year. This means that jobs with extra risks of 0.00001 fatality per worker per year pay $.0352 per week more than jobs with no such risk. This is equal to $1.76 per year.

If 100,000 workers will accept $1.76 per year to incur an additional on-the-job mortality risk of 1 in 100,000 per year, then together they will accept $176,000 per year as compensation for the expected loss of one life per year (or one "statistical" life as commonly stated in the literature). Similarly, workers who accept a wage rate of $.0352 per week less than other workers, and are exposed to a loss of life probability 0.00001 per year less than other workers, can be said to be willing to pay $176,000 per year to save one statistical life.

Tables 2.10 and 2.11 show the data used by Thaler and Rosen to estimate their wage risk function, and Table 2.12 shows the results for four different functional forms of the wage risk function.

From these equations four different values of a statistical life are obtained. These are shown in Table 2.13.

Table 2.10

Thaler and Rosen (1976) Risk Data on
Extra Deaths by Occupation

Occupation	Risk*	Occupation	Risk*
Fishermen	19	Truck drivers	98
Foresters	22	Bartenders	176
Teamsters	114	Cooks	132
Lumbermen	256	Firemen	44
Mine operatives	176	Guards, watchmen, and doorkeepers	267
Metal filers, grinders and polishers	41	Marshals, constables, sheriffs and bailiffs	181
Boilermakers	230	Police and detectives	78
Cranemen and derrickmen	147	Longshoremen and stevedores	101
Factory painters	81	Actors	73
Other painters	46	Railroad conductors	203
Electricians	93	Ships' officers	156
Railroad brakemen	88	Hucksters and peddlers	76
Structural iron workers	204	Linemen and servicemen	2
Locomotive firemen	186	Road machine operators	103
Power plant operatives	6	Elevator operators	188
Sailors and deckhands	163	Laundry operatives	126
Sawyers	133	Waiters	134
Switchmen	152		
Taxicab drivers	182		

Source: Society of Actuaries

* Units of measure are extra deaths per 100,000 policy years. To convert to the probability of an extra death per year on each job multiply by 0.00001.

Table 2.11

Variables Used in Thaler and Rosen (1976)

Variable	Mean	Standard Deviation
Dummy variables		
Urban	.69	.46
Northeast	.28	.45
South	.29	.45
West	.17	.38
Family size exceeds 2	.76	.42
Manufacturing industry	.24	.42
Service industry	.58	.49
Worker is white	.90	.30
Worker is employed full time	.98	.10
Worker belongs to union	.45	.49
Worker is married	.92	.26
Occupation is operative	.27	.44
Occupation is service	.45	.49
Occupation is laborer	.22	.42
Continuous variables		
Age (years)	41.8	11.3
Education (years)	10.11	2.73
Weeks worked in 1966	49.4	5.4
Hours worked last week	44.9	11.6
Risk (probability x 10^5)	109.8	67.6
Weekly wage (week prior to survey)	$132.65	50.80

* Mean is proportion in sample with designated characteristic. The number of observations is 907.

Table 2.12
Thaler and Rosen (1976) Regression Estimates - Linear Form

Independent Variable	Equation 1	Equation 2	Equation 3	Equation 4
Risk	.0352	.0520	.100	.0410
	(.0210)	(.0219)	(.108)	(.102)
Risk x age	-	-	-.0019	-.0030
			(.0018)	(.0019)
Risk x married	-	-	.0791	.0701
			(.0380)	(.0412)
Risk x union	-	-	.0808	.0869
			(.040)	(.042)
Risk x white	-	-	-.118	-
			(.072)	
Urban	13.80	15.71	17.0	17.0
	(4.2)	(2.95)	(3.0)	(3.2)
Northeast	-3.71	-4.29	-4.27	-4.92
	(3.65)	(3.67)	(3.63)	(3.83)
South	-8.86	-8.90	-10.5	-8.18
	(3.70)	(3.74)	(3.72)	(3.97)
West	9.13	10.30	9.57	9.50
	(4.13)	(4.18)	(4.12)	(4.37)
Age	3.89	3.81	3.83	3.78
	(0.80)	(0.83)	(0.82)	(0.87)
(Age)2	-.0479	-.0468	-.0442	-.0415
	(.0092)	(.0097)	(.010)	(.011)
Education	3.40	3.27	4.13	4.81
	(0.55)	(2.40)	(2.39)	(2.80)
(Education)2	-	-.021	-.0237	-.042
		(.128)	(.128)	(.148)
Manufacturing industry	-	-	-13.0	-14.7
			(4.3)	(4.62)
Service industry	-	-	-9.45	-10.9
			(3.95)	(4.24)
White	22.92	22.93	37.7	-
	(4.53)	(4.50)	(9.6)	
Family size > 2	-	-	.400	2.10
			(3.57)	(3.89)
Union	25.5	27.16	15.9	15.39
	(3.25)	(3.23)	(5.4)	(5.72)
Full-time	-1.63	-.86	-1.16	.45
	(12.9)	(12.6)	(12.6)	(15.0)
Hours worked	1.50	1.41	1.47	1.44
	(.12)	(.12)	(.123)	(.129)
Occupation 1: operative	-18.7	-	-13.9	-13.5
	(9.2)		(3.24)	(3.51)
Occupation 2: service worker	-24.6	-	-18.1	-19.9
	(9.5)		(4.66)	(5.05)
Occupation 3: laborer	-25.0	-	-	-
	(13.4)			
SES 1	-	4.68	-	-
		(5.17)		
SES 2	-	-17.17	-	-
		3.34)		
SES 3	-	-20.69	-	-
		(5.53)		
R^2	.41	.41	.42	.39
Number of observations	907	907	907	813
Sample	All	All	All	White only

Note: The dependent variable is the weekly wage rate. The SES index has been converted to dummy variables. Standard errors are in parentheses.

Table 2.13

Estimated Values for a Statistical Life
Thaler and Rosen (1976)
(1967 U.S. Dollars)

Equation 1	$176,000
Equation 2	260,000
Equation 3	117,500
Equation 4	96,000

Note: The calculations utilized all of the coefficients, significant and not significant.

Thaler and Rosen also estimated a semilog linear version of the wage risk equation. This gave systematically lower estimates for the value of a statistical life.

Violette and Chestnut review nine studies, including Thaler and Rosen's, that employ basically the same methodology as described above. They point out that in virtually all of these studies, the risk variable seems a significant determinant of wage rates. A summary of the empirical results of these studies is given in Table 2.14. The table shows that judgemental-best estimates of the value per statistical life derived from studies using variants of the same methodology vary by more than an order of magnitude. This is a disturbingly high range due, in part, to the following issues and controversies identified by Violette and Chestnut:

i) The potential for omitted variable bias. In any use of regression analysis there is the danger that the omission of explanatory variables will bias the estimate of those variables included. This is a problem when the omitted variable(s) are systematically related to the variables included. Otherwise the omission of important variables only affects the confidence intervals (i.e., the standard errors) of the included variables.

ii) The potential biases in the job-risk data sets. Lipsey (1976) commented

Table 2.14

Estimates of the Marginal Willingness to Pay for Reductions in Risks
(expressed in May 1982 dollars)[a]

	Study	Mean Risk Level for the Sample[b]	Value of Reducing Risk of Death by 1 Chance in 10,000[c]		Value per Statistical Life[c]	
			All Estimates	Judgemental Best Estimates[d]	All Estimates	Judgemental Best Estimates[d]
1.	R. Smith (1974)	1.0 to 1.5	$750 1,320	($750)	$7.5 x 10^6 13.2 x 10^6	(7.5 x 10^6)
2a.	Thaler and Rosen (1975) w/o risk interaction terms[e]	11.0	$39 50 54 75	($ 57)	$3.90 x 10^5 5.05 x 10^5 5.42 x 10^5 7.46 x 10^5	(5.7 x 10^5)
b.	with risk interaction terms[f]	11.0	$1.40 $20 $27 $34		$ .14 x 10^5 1.99 x 10^5 2.75 x 10^5 3.36 x 10^5	
3.	R. Smith (1976)	1.0 to 1.5	$322 344	($333)	$3.22 x 10^6 3.44 x 10^6	(3.3 x 10^6)
4.	S. K. Viscusi (1978b)	1.2	$146 223 266 344 395 421 438	($390)	1.46 x 10^6 2.23 x 10^6 2.66 x 10^6 3.44 x 10^6 3.95 x 10^6 4.21 x 10^6 4.38 x 10^6	(3.9 x 10^6)
5.	A. Dillingham (1979)	1.7	$34 40 41 43 110	($40)	3.4 x 10^5 4.0 x 10^5 4.1 x 10^5 4.3 x 10^5 11.0 x 10^5	(4.0 x 10^5)
6.	C. Olson (1981)	1.0	$760	($760)	7.1 x 10^6	(7.1 x 10^6)
7.	V. K. Smith (1982)	3.0[g]	$73 to $207[h]	($100)	$7.3 x 10^5 to 20.7 x 10^5	(1.0 x 10^6)
8.	Arnould and Nichols (1983)	11.0	$64	($64)	6.40 x 10^5	(6.40 x 10^5)

Table 2.14

Notes

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a Adjustments to 1982 dollars were made by using the consumer price indices for all items published by Council on Economic Advisors, Economic Indicators, December 1982.

b The risk level is expressed in annual deaths per 10,000 workers. The value should be viewed as an approximate figure since many of the studies reported risks in different units requiring transformation to common units.

c The multiple estimates are derived from the different model specifications examined. It was felt that presenting estimates from all the specifications is better than simply showing the range of estimates since one outlier can distort the range.

d The judgemental "best" estimate represents ant model specifications examined. It was felt that presenting estimates from all the specifications is better than simply showing the range of estimates since one outlier can distort the range.

d The judgemental "best" estimate represents a guess by these reviewers based on judgement and the information provided by the authors of each study.

e These estimates are from Thaler and Rosen (1975) equations that did not include interaction terms between risk and other variables.

f From specifications including interactions variables between risk and nonrisk variables.

g Calculated by assuming that .4 percent of all injuries are fatal. This is ratio for the BLS injury statistics is reported by Viscusi (1978b, p. 365).

h Calculated assuming the risk premium for risks of fatal injuries ranged from 33 percent to 100 percent of the premium associated with all risks.

Source: Violette and Chestnut (1983)

that the job-risk data set used by Thaler and Rosen reflect all risks to life whether or not they are job related. Hence, their data set does not necessarily show the risks related to the occupations themselves, but rather to the people involved in the occupations.

The other major source of job-risk data used in several of the other studies noted in Table 2.14 is the U.S. Bureau of Labour Statistics. The Bureau provides risk data by industry rather than occupation so that differential risk within industries is neglected. Landefeld and Seskin (1982) show that this can lead to serious overstatement of the value of a statistical life.

Unfortunately, there is a paucity of appropriate risk data sets so that these deficiencies cannot be easily remedied.

iii) Specification of the correct functional form for the wage risk function. The value of the estimated risk coefficient and the corresponding value of a statistical life can be significantly affected by the specification of the functional form. Traditional economic theory, as represented by Figure 6, is inadequate to determine what the appropriate functional form should be except to the extent that there is a positive relationship between wage rate and risk.

iv) The functioning of the labour market with respect to risk. The wage risk analysis in all the studies implicitly assumes that the labour market is in equilibrium, that labour is mobile and can move from job to job in response to changing wage/risk options, and that the lack of information about job risks does not bias the risk coefficient estimates.

v) The separation of the estimated wage premiums into compensation for risks of fatal accidents and compensation for nonfatal injuries. Available information on fatal accidents and nonfatal injuries is generally inadequate to determine their respective contributions to wage differentials. The importance of occupational health risks is even more difficult to ascertain. Where risks of fatalities have been separated out, as in most of the studies cited in Table 2.14, it has been

accomplished through somewhat arbitrary procedures.

vi) The possible inadequacy of wage rates as a measure of worker compensation. Worker compensation can include nonwage fringe benefits, workmen's compensation, disability insurance, life insurance, as well as wages. Wage rate differentials are, therefore, unlikely to capture the full extent of compensation for incurring increased risk and this will tend to undervalue statistical lives.

In view of all these issues the variation in estimates of the value of a statistical life obtained using labour market data may not be surprising. Further work, using more appropriate data may yet resolve a good deal of the discrepancies.

The two other approaches mentioned earlier for estimating the value of a statistical life: consumer market studies and contingent market approaches, merit some attention. Again, Violette and Chestnut (1983) provide a useful summary of this work.

Consumer market studies are similar in principle to the labour market studies. Implicit welfare accounting values of statistical lives are estimated from an analysis of consumer market data. The results of three such studies are summarized in Table 2.15.

While they avoid the use of suspect labour market data, these studies involve problems of their own. Smoke detectors, studied by Dardis (1980), were a new technology making their entrance into the market at the time he studied them. Their prices were falling dramatically and people were learning about the existence of the product. These factors complicate the interpretation of the data. Furthermore, smoke detectors protect property as well as human life and there is no obvious basis for determining the relative importance of each.

Blomquist (1977) examined people's willingness to take the time and incur the inconvenience of using their seat belts to reduce the risk of injury and death. Despite his somewhat ingenious methodology, the problem of valuing

Table 2.15

Summary of Value of Life Estimates from Consumer Behavior Studies

Study	Initial Risk Level	Increment of Risk	Average Value Per Life	Value Per Life 1982 Dollars	Nature of Risk Examined
Dardis (1976$)	8.77×10^{-5}	3.16×10^{-5}	$189,049 to $294,968	$318,334 to $496,688	Residential fire fatalities
Blomquist (1972$)	3.027×10^{-4}	1.514×10^{-4}	$236,000	$340,747	Automobile accident fatalities
Ghosh, Lees and Seal (1973$)	Not reported	Not reported	$230,394	$496,966	Automobile accider fatalities on British motorways

Source: Violette and Chestnut (1983)

time saved confounds his estimates. His use of a portion (30 - 50%) of the wage rate for this purpose is not uncommon in economic studies but it is problematic. Do people really value very small increments of time saved that cannot be readily transformed into income? Other difficulties include the estimated time taken to fasten a seat belt and the assumption that people are well informed about the safety benefits from using seat belts.

In the study by Ghosh, Lees and Seal (1975) a relationship is derived between the optimal speed of travel on British freeways and values of life and time. While it may be true that people trade off travel time and the probability of injury or death, there is also the pleasure of travelling at increased speed. This suggests that Ghosh, Lees and Seal underestimated the value of a statistical life. Moreover, as with the Blomquist study, the results obtained depend critically on the accuracy of the assumed value of travel time, which itself is the subject of a whole literature and research effort.

Table 2.16 summarizes the results of several contingent market studies reviewed by Violette and Chestnut (1983). Two aspects are significant. Firstly, the enormous range in values for a statistical life and, secondly, the possible relationship between these values and a combination of the initial level of risk and the increment of risk considered.

Considerable work has been undertaken in the past few years on contingent value studies, in a variety of settings. (Brookshire et al 1981). Much has been learned about questionnaire design and administration to minimize the problem of asking people to respond to hypothetical questions. However, the studies referred to in Table 2.16 have not benefited from this recent work and so the results obtained can only be regarded as highly tentative.

2.3.2.4 Canadian Estimates of the Value of Reduced Risk of Injury

Hinton (1980) obtained two matched-data series on average weekly wages and nonfatal accident frequencies (temporary total disability accidents per million man-hours) from the Ontario Workmen's Compensation Board (OWCB). The data covered 108 rate-number employer groups for each year from 1975 to 1978. These OWCB groups are a mix of an occupational and an industrial classification designed to correspond to common levels of hazard. As such,

Table 2.16

Summary of Value of Life Estimates from Contingent Market Studies[1]

Study (Year Dollars)	Initial level of risk	Increment of risk	Average value per life (thousands)	May 1982 $ (thousands)	Nature of risk examined
Acton (1972)	4×10^{-3}	2×10^{-3}	$28	$64	heart attack fatalities
	4×10^{-3}	10^{-3}	$43	$98	
	2×10^{-2}	10^{-2}	$7.4	$17	
	2×10^{-2}	5×10^{-3}	$12	$27	
Jones-Lee (1975)	0 to 4×10^{-5}	2×10^{-6} to 2×10^{-5}	$178 to $27,750 (mean = $6,700)	$317 to $49,423 (mean = $5,343)	airline accident fatalities
Murphy[2] (1978)	—	—	$2,000	$2,937	—
Frankel (1979)	1.5×10^{-6}	1.5×10^{-6}	$2,970	$3,922	airline accident fatalities
	10^{-4}	10^{-4}	$50	$60	
Mulligan[3] (1977)	10^{-4}	9×10^{-4}	$45	$71	nuclear plant accident injuries
	10^{-5}	9×10^{-5}	$315	$498	
	10^{-6}	9×10^{-6}	$2,627	$4,151	
	10^{-7}	9×10^{-7}	$22,000	$34,760	
	10^{-8}	9×10^{-8}	$204,000	$322,320	

1 This table is for summary and comparison purposes. Before using the estimates, the reader should understand the assumptions and procedures by which they were obtained.

2 This estimate was not linked to a specific risk or risk increment although the author states that it is relevant only for small changes in risks.

3 These are based on the assumption that the survey question referred to annual risks and that all injuries are fatal.

Note: The original version of this table in Violette and Chestnut contained numerous typographical errors. These have been corrected in the above table.

Source: Violette and Chestnut (1983)

the categories correspond reasonably well to the equal risk concept of an occupation used in Hinton's model.

Using these data, Hinton estimated the following equation:

$$W_{it} = V_t + dR_{it} + e_{it} \qquad 9.$$

Where: W_{it} is the average wage in employment group i and time t

V_t is a time varying constant

R_{it} is the accident frequency rate in employment group i at time t

e_{it} is an error term

Hinton's results derived from a pooled, data set are given in the second column of Table 2.17. Results are also shown for two other specifications of the wage-risk equation. In all specifications the risk coefficient is positive, and statistically significant at the 0.01 level for a one-tail test in the linear and polynomial versions.

The estimated value of a statistical accident based on the results in Table 2.17 is $5,000 for the linear equation and $12,000 for the polynomial equation. Hinton presents these results as $8,500 + 3,500. After adjustments for the exchange rate and inflation, this is very close to Viscusi's (1978) estimate using U.S. data of $8,770 Canadian. Hinton concludes that "more work is required in this area if we are to develop a representative schedule of values of 'life and limb' to facilitate policy analysis in the field of occupational health and safety".

2.3.3 Adjusted Willingness to Pay/Human Capital Approach-- The Value of Saving a Life

Landefeld and Seskin (1982) use 1977 data for the U.S.A. to compare the value of saving a life as estimated by the standard human capital approach and their own recommended adjusted WTP/HK approach. The results are shown in Table 2.18.

Table 2.17

Regression Estimates

Independent Variables	Coefficients[a] (and Standard Errors[b])		
	Linear	Polynomial	Semilog
Risk	0.20599**	0.48697**	0.000347*
	(0.0654)	(0.18472)	(0.000212)
Risk Squared	-	-0.0009*	-
	-	(0.000554)	-
Dummy 1976	24.31**	23.75**	0.1109**
	(1.6942)	(1.7244)	(0.0055)
Dummy 1977	45.03**	44.26**	0.1950**
	(1.6882)	(1.7484)	(0.0055)
Dummy 1978	61.38**	60.74**	0.2574**
	(1.7269)	(1.767)	(0.0056)
R^2	0.83	0.83	0.89
SEE	12.26	12.23	0.0397
n	432	432	432

Note: a** denotes significance, according to a one-tailed test at the 0.01 level, where the critical t-value is 2.326

* denotes significance, according to a one-tailed test at the 0.10 level, where the critical t-value is 1.282

b the standard errors, in parenthesis, are corrected for the 108 degrees of freedom accounted for by the fixed-effects terms.

Source: Hinton (1980)

Table 2.18

Alternative Estimates of the Value of Life, 1977
(1977 dollars U.S.)

Age Group (years)	Adjusted Willingness-to-Pay/ Human Capital[a]		Standard Human Capital[b]	
	Male	Female	Male	Female
0 to 1	668,461	457,139	31,918	28,625
1 to 4	704,303	481,290	39,657	35,539
5 to 9	770,438	526,147	58,367	52,273
10 to 14	850,562	580,490	89,604	80,196
15 to 19	928,875	623,496	130,874	112,390
20 to 24	976,304	626,792	170,707	133,238
25 to 29	966,434	586,710	196,612	136,664
30 to 34	880,836	526,912	205,062	130,044
35 to 39	790,452	465,115	197,881	121,547
40 to 44	660,193	414,562	180,352	111,647
45 to 49	522,064	332,221	156,297	99,796
50 to 54	380,389	266,482	124,989	86,286
55 to 59	240,382	201,726	86,246	70,417
60 to 64	119,328	143,086	45,169	53,426
65 to 69	50,127	99,056	18,825	39,213
70 to 74	25,294	69,306	9,781	29,189
75 to 79	12,816	48,202	5,108	21,728
80 to 84	6,787	33,936	2,820	16,787
85+	2,039	9,966	943	5,705

[a] Adjusted willingness-to-pay/human capital estimates based on the present value of both expected lifetime after-tax income and housekeeping services (where income is estimated from earnings by using the ratio of disposable income to wages and salaries equal to 1.33); an after-tax real rate of return equal to 3 per cent; an annual increase in labor productivity of 1 per cent; and a risk-aversion premium of 1.6.

[b] Standard human capital estimates based on the present value of both expected lifetime earnings and housekeeping services: a real discount rate of 10 per cent; and an annual increase in labor productivity of 1 per cent.

Source: Landefeld and Seskin (1982)

A major reason for the consistently larger adjusted WTP/HK estimates than the standard HK approach is the use of the lower discount rate. If a discount rate of 3% is used in the standard HK approach, the value of life for males aged 40-44 rises to one-half of the adjusted WTP/HK estimate.

2.3.4 Estimating the Value of the Health Benefits from Automobile Emissions Control

Several studies provide estimates of the value of health benefits from automobile emissions control, though none of them make use of the most recent work on output or welfare accounting. These studies are reviewed and then a specific strategy is recommended.

2.3.4.1 A Review of Studies in Which Health Benefits from Automobile Emissions Control Have Been Estimated

In 1974 the U.S. Senate Committee of Public Works published a report on Air Quality and Automobile Emission Control (U.S. Senate 1974). This extensive report gives a detailed assessment of the economic benefits of air quality improvements. Effects of air quality on health, vegetation, and materials are all considered. In addition, the proposition that the value of improved air quality can be estimated through analysing property and labour market differentials is also considered, and some empirical results are presented.

The sections in the report that deal specifically with health impact evaluation were written in advance of most of the work that has provided the basis for the present report. Nevertheless, they remain helpful despite some important inadequacies. The first of these is the way in which morbidity impacts were valued. A range of $1-$10/person day of discomfort was arbitrarily selected. This is said to be consistent with values used in another study, but no methodological or empirical basis is provided. Impacts on mortality were valued at $200,000/life and Thaler and Rosen (1976) is one of two references cited in support.

The health-impact values derived from these morbidity and mortality costs were then added directly to the air-quality improvement values that were independently estimated from an analysis of the property market. This raises

a potentially serious problem of double-counting since one of the reasons for lower property values in polluted areas is likely to be people's concerns over effects on health, in which case the same effect is being counted twice.

The authors of the U.S. Senate report argue that "when considering major (nonmarginal) changes in ambient standards, there is the danger that benefits derived from market data (i.e., property or labour markets) underestimate the total value of the benefits to the recipients. Adding the benefits from the specific cost studies to the market based benefits should compensate somewhat for this underestimation" (p.180). The problem with this strategy is that the result is conceptually unsound and the health impact estimates are inaccurate to an unknown degree, and in an unknown direction.

David Harrison (1975) published a book that analysed the cost and benefit distribution of Federal (U.S.) automobile emission controls. In his study he recognized the desirability of placing a dollar value on the benefits to show the willingness to pay of various groups for air quality benefits. However, he eschewed the use of such dollar values on the grounds that sufficiently detailed willingness to pay estimates by household groups were unavailable.

A study by Small (1977) provides rough and aggregated measures of the economic costs and benefits imposed on society by air pollution from various transport modes in urban areas. The estimated value of health impacts was based on Lave and Seskin (1970), which utilized the output accounting values from Rice (1966).

In a book devoted entirely to air pollution and human health, Lave and Seskin (1977) updated their earlier work but maintained their use of output accounting values for health impacts by using values from Cooper and Rice (1976). Commenting on the possibility of adopting a welfare accounting approach (which they refer to as an implicit value approach) they say "...although we regard the Thaler and Rosen estimates as worthy of consideration, we are somewhat reluctant to use their figures for two reasons. First, the foregone-earnings (i.e., output accounting) approach has the virtue of giving numerical estimates that are indisputable measures of what they say they are. Second, there is good reason to expect foregone

earnings to underestimate the true value of health and life. Since we have elected to maintain a conservative posture in estimating the air pollution-mortality association by using estimates at the bottom range of those stemming from our work, we shall continue this position by selecting conservative figures for the value of life (and health)" (p.220).

Southworth (1979) developed a household utility maximization model in which the benefits from automobile emissions control take the form of decreased costs for certain goods (e.g., medical treatment and house maintenance) and decreased mortality. He evaluated the nonrisk health benefits of control by combining average wage rates, average hospitalization costs, and the expected change in mortality. For this purpose he assumed that morbidity is correlated with mortality.

In addition to these benefits, Southworth drew on the results of Thaler and Rosen (1976) to allow for the benefits to households of reduced risks of mortality, morbidity, and other costs from automobile pollution. He showed that this justified a greater degree of emissions control than had he relied exclusively on the results of Cooper and Rice (1976) to estimate the health impact benefits.

Finally, the work of Richard Schwing and his colleagues at General Motors deserves comment. A paper first delivered at a conference in 1976 was published, with minor revisions in 1980 (Schwing et al 1980). This paper provides a benefit-cost analysis of the automotive emission reductions required by the U.S. Clean Air Act of 1970.

Health impacts were valued using the output accounting approach, with specific values drawn from Rice (1966). Apparently, Schwing et al had completed their study prior to the publication of Cooper and Rice (1976), though there would seem to have been ample time for revisions before the published version of their paper was released. One peculiarity of Schwing et al's use of Rice's results is that, despite allowing for differences in air pollution impacts on various population groups (white males, nonwhite males, white females and nonwhite females), all of the impacts were valued as if they had been incurred by white males. Within the context of the output

accounting approach, this will overestimate the health benefits obtained from cleaner air since the earnings of this group exceeds that of the others.

A potentially significant error in the opposite direction was made by Schwing et al in assuming that nonmarket benefits from reduced air pollution are only twice as great as the market benefits. Schwing et al note that these nonmarket benefits include reduced pain and suffering, diminished sensory discomfort and annoyance, and increased pleasure or satisfaction derived from the enjoyment of a cleaner environment. A rough comparison of the value of health benefits alone estimated by welfare accounting methods and output accounting methods suggests a 10-fold difference is more reasonable. (Compare the cost/mortality from Cooper and Rice, Table 2.8, of about $70,000 in 1982 dollars, with the values of a statistical life in Table 2.13).

The relevance of the above suggested discrepancy may be questioned on the grounds that most of the health benefits from automotive emissions control are expected to come primarily from reduced morbidity rather than reduced mortality (Kolemeychuk et al 1983). Since most of the welfare accounting studies have focussed on mortality risks, no simple comparison between welfare and output accounting values or reduced morbidity is currently possible.

This fact raises the more general question of the use that can be made of the existing welfare accounting estimates of health benefits in assessing the desirability of more stringent automobile emissions regulations in Canada. At present, an inadequate distinction is made in the extant studies between mortality and mobidity risks. The attempts that have been made tend to be arbitrary. Contingent valuation, using carefully designed questionnnaires, offers the most promising avenue for separating the various kinds of risks and attaching values to them. However, the existing contingent valuation studies are too unreliable to be of use in this regard.

The approach taken by Lave and Seskin (see above quotation) to health benefit evaluation is certainly viable. Providing one does not use the results without qualification and discussion in a benefit-cost analysis, output accounting may provide some economic estimates that are useful for

decision-making purposes. If this is done, it will be essential to emphasize the extent to which these estimated costs diverge from the appropriate welfare accounting costs (measured as WTP or WTA). Furthermore, in all of this analysis the distributional aspects of the estimates should be considered.

While it is recommended that the above research proceed, there is another overriding argument to be considered. Public policy directed at risk reduction can be greatly assisted by calculation of the costs per life saved and illness/injury avoided per dollar expended in various programs. If desired, risk categories can be further distinguished according to the nature of the risk: e.g., voluntary and involuntary risks, size of group at risk, small and large risks, short-term and long-term risks. Within the chosen set of categories, efficient public policy requires an equality of risk reduction per (marginal) dollar spent. If this equality does not hold, then risk can be reduced merely by reallocating funds among programs.

Estimates of the costs of controlling automobile emissions can then be compared on a risk reduction per dollar basis, with other activities in the same risk category. If emissions control offers a cheaper way of reducing the relevant risks than prevailing programs, then a good case can be made for the regulation coupled with a relaxation of the less cost-effective programs. This may shift the burden of the risks and, if so, the distributional implication must be judged acceptable to determine the overall acceptability of this proposal.

Estimates of the value of risk reduction are irrelevant to the above process. They are only necessary to decide whether the total effort devoted to risk reducton are, in some sense, optimal. In light of the lack of reliable, off-the-shelf estimates, and the difficulty of making good estimates given the present state of the art, useful economic input to this issue will remain elusive for years to come.

2.3.4.2 An Approach to Estimating the Value of Health Impacts from Exposure to Emissions from Automobiles

In this section an attempt is made to outline a practical approach for estimating the value of a reduction in health impacts associated with the further control of automotive emissions. Despite the theoretical appeal of welfare accounting it is proposed that an output accounting framework be adopted. The reasons for this are:

i) The practicality of the approach and lack of ambiguity in the results.

ii) The irrelevance of most of the extant welfare accounting estimates of the value of health risks to this specific issue. (Most of the welfare accounting studies deal with mortality, whereas morbidity is the central concern with respect to automotive emissions).

iii) Output accounting estimates can be interpreted as a lower bound of the welfare accounting values.

This is the same approach as recommended by the US Environmental Protection Agency: "Morbidity (illness) should be valued by estimating its direct cost, unless it is feasible to use willingness to pay measures." (US EPA 1983)

It is beyond the terms of reference for this study to generate a comprehensive set of health impact estimates. However, it is possible to report on the availability of Canadian data for making such estimates and to provide some of the relevant information. The categories of costs that can be estimated with some accuracy are: medical costs, the value of lost time in paid employment, and the value of lost household work. Each of these is discussed below.

Medical Costs

Medical costs can be divided into several categories:

- nonhospitalized treatment:
  - physician's services
  - diagnostic services
  - drugs (prescription and nonprescription)

- hospitalized (and other institutional) treatment:
  - physician's services
  - hospital services (including accommodation, nursing, diagnostic testing and drugs)

There is no central source of information on these costs in Canada although the various provincial health care systems and hospital associations collect a significant portion of the requisite data. The problem is that the medical cost information that is routinely published is not usually broken down by diagnostic category. This makes it difficult to relate medical costs to specific diseases, such as those listed in Table 2.19. (The diagnostic categories most relevant to the health impacts from automotive emissions are shown in column two of Table 2.19. The International Classification of Diseases, Ninth Revision, has been used for this purpose).

Table 2.20 shows the results of a preliminary attempt to estimate the costs of medical treatment associated with individual cases of the diseases identified in Table 2.19. The hospital costs for males and females are based on Barer's (1981) estimates, suitably adjusted for inflation using the health cost component of the consumer price index. Since Barer's costs only cover those borne directly by the hospitals, it was necessary to add in an estimate of the physicians' costs. These were obtained from the Saskatchewan Medical Care Insurance Commission (MCIC) for the year 1982/83.

MCIC also provided information on the cost of nonhospital treatment by physicians for each of the relevant diagnostic categories, as shown in the fourth and fifth columns of Table 2.20. The costs in Table 2.20 also include an estimate of the prescription drug costs derived from Fraser, Spasoff and Prime (1976) and an amount for diagnostic services (15% of the physicians' costs, based on general information given in the MCIC annual report for 1983).

It is stressed that the cost estimates given in Table 2.20 are highly tentative. Although a deliberate effort was made to avoid double counting and significant omissions, the combination of cost figures from different sources, different periods of time, and different regions is vulnerable to

Table 2.19

Summary of Possible Major Effects of Automotive Emissions on Health

Substance/Effect[1]	ICD9 Code	Susceptible Population[3]	Severity of Effect[2] Treatment Not Required	Visit to Doctor	Hospitalization Required
Carbon Monoxide					
1. Myocardial infarction (heart attack)	410	People with cardiovascular disease			x
2. Aggravation of angina pectoris (attacks of sharp chest pain due to inadequate blood supply)	413	As above	x	x	x
3. Intermittent claudication (episodes of leg pain or limping due to inadequate blood supply)	411	People with peripheral vascular disease	x	x	
4. Impaired vigilance (visual, auditory, reaction time, time estimation, etc.)	781	General population	x		
5. Effect on fetal development (decreased birth weight, increased perinatal deaths, and decreased weight gain in newborns; reduced learned and general abilities in later childhood)[4]	319 635 646 647 648 653 654	Pregnant women	x	x	x

Substance/Effect	ICD9 Code	Susceptible Population	Severity of Effect[2] Treatment Not Required	Visit to Doctor	Hospitalization Required
Nitrogen Dioxide					
6. Increased incidence of acute respiratory illness	460 466	Very young children	x	x	
7. Aggravation of respiratory disease (attacks of asthma, bronchitis, or emphysema)	490- 493	People with asthma, chronic bronchitis, or emphysema)	x	x	
8. Impaired lung function and likelihood of incidence of respiratory symptoms (chest discomfort, cough, shortness of breath)	443 478	General population			
Ozone					
9. Increased incidence of discomfort and respiratory symptoms (chest discomfort, cough, headache, sore throat, eye irritation, shortness of breath)	379 443 478	General population	x	x	
10. Aggravation of respiratory disease (same as NO_2)	490- 493	People with asthma, chronic bronchitis or emphysema	x	x	x
11. Decreased resistance to infection	460 466 480 487	General population	x	x	x

Notes to Table 2.19

1. Both morbidity and mortality are possible, but most of the health benefits from automotive emission control are expected to come from reduced morbidity rather than reduced mortality.

2. In virtually all cases for CO and NO_x and most cases for O_3 the effects during the highest ambient concentrations experienced in Canada will be at the low end of the severity scale. It is unlikely, except in the case of peak concentrations of O_3, that hospitalization would be required for people suffering the effects listed in the table.

3. Where the susceptible population is defined as the general population, not everybody will suffer the effect at the highest concentrations of the pollutants that are experienced in Canada. Only a fraction--the most sensitive of those that are exposed to the highest concentrations--will experience the effects.

4. Ambient CO levels in Canada are very unlikely to reach levels high enough so that Carboxyhaemoglobin levels reach the point where these effects will occur. Only those pregnant women that are heavy smokers would be at risk.

Table 2.20

ESTIMATED MEDICAL COSTS BY DIAGNOSTIC CATEGORY

ICD9 Code	Hospital Cost/Case Male (1983$)	Hospital Cost/Case Female (1983$)	Nonhospital Cost/Case Male (1983$)	Nonhospital Cost/Case Female (1983$)
319	1650	1768	76	103
379	1641	1612	47	47
410	1736	1709	63	56
411	1305	1322	58	68
413	1400	1385	64	59
443	1318	1278	54	47
460	966	961	34	34
466	1001	995	41	40
478	2023	2014	40	38
480	1532	1531	43	38
487	1004	1009	38	38
490	1266	1253	42	41
491	1279	1266	52	46
492	1317	1305	62	65
493	1260	1273	87	86
635	na	1070	na	115
646	na	889	na	36
647	na	1490	na	*
648	na	1619	na	72
653	na	1559	na	102
654	na	1562	na	67
781	1113	1109	45	44
787	1117	1114	40	37
788	1127	1143	42	41

na - not applicable

* - no data

Sources: Saskatchewan Medical Care Insurance Commission
Fraser, Spasoff, Prime (1976)
Barer (1981)

these types of errors. Nevertheless, the table does show the variation that exists in medical costs among diseases and especially between hospital and nonhospital treatments. Differences between males and females may not be significant since they may reflect differences in the ages of the patients, rather than differences in sex. Clarification of this issue would require a more detailed analysis than that undertaken for this report.

Lost Time in Paid Employment

The value of output lost through interruption in the supply of labour is customarily valued at the pre-tax wage rate. The basis for this is the marginal productivity theory of wages, one result of which is that employers will set the size of their labour force such that the value of the output of the marginal employee is just equal to the wage rate. For various reasons this is only an approximation. However, it does provide a simple way of estimating the value of lost time in paid employment due to ill health. Information on wages and salaries is gathered routinely by Statistics Canada, though only occasionally is it published according to age and sex. (Statistics Canada 1981). A further breakdown by occupation is available from census data but requires an analysis specifically for this purpose.

The extent to which ill health causes time off from paid employment has not been thoroughly researched in Canada. Perhaps the only source of information is the Health Survey of Canadians (1981) which was originally intended to be the first of an ongoing series of annual surveys. Lack of funds has not permitted this and so the information gathered from July 1978 to March 1979 is all that is presently available for estimating the impact of ill health on people's work activity.

Table 2.21, which is reproduced from the survey report, shows the kind of information which is available. Further disaggregation by age and sex is possible, but the sample size is such that in many cases the reliability of the estimates becomes unacceptable. Dividing the number of disability days (i.e., bed-days, major activity-loss days and cut-down days) by the number of health problems of each type, gives an estimate of the short-term disability caused by type of health problem.

Table 2.21

Prevalence of Health Problems by Selected Health Behaviour, by Type of Health Problem, Canada, 1978-79(1)

Type of health problem		Total population			Disability days		Consultations		Drug use		Activity limitation		None of these	
		Both sexes	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
		in thousands												
Total population(2)	No.	23,023	11,417	11,606	1,111	1,654	2,086	3,031	4,658	6,363	1,250	1,416	5,405	3,989
	%	100.0	49.6	50.4	4.8	7.2	9.1	13.2	20.2	27.6	5.4	6.2	23.5	17.3
At least one problem	No	12,510	5,714	6,796	1,110	1,647	1,723	2,556	3,254	4,776	1,250	1,416	1,359	1,049
	%	100.0	45.7	54.3	8.9	13.2	13.8	20.4	26.0	38.2	10.0	11.3	10.9	8.4
No problem	No	10,513	5,703	4,811	--	--	363	475	1,404	1,587	-	-	4,046	2,940
	%	100.0	54.2	45.8	--	--	3.5	4.5	13.4	15.1	-	-	38.5	28.0
Health problems														
Total problems	No.	25,526	10,558	14,967	1,115	1,671	1,380	1,964	2,474	4,299	1,247	1,415	5,724	7,702
	%	100.0	41.4	58.6	4.4	6.5	5.4	7.7	9.7	16.8	4.9	5.5	22.4	30.2
Mental disorders	No	1,000	363	637	18	53	43	72	235	501	49	77	70	74
	%	100.0	36.3	63.7	1.8	5.3	4.4	7.2	23.5	50.1	4.9	7.7	7.1	7.4
Diabetes	No	379	149	230	--	--	--	18	59	104	15	25	79	107
	%	100.0	39.2	60.8	--	-	-	4.8	15.6	27.3	3.9	6.7	20.9	28.3
Thyroid disorders	No	297	41	256	-	--	-	14	20	119	1	-	21	129
	%	100.0	13.7	86.3	-	--	-	4.8	6.8	40.2	2	--	6.9	43.3
Anemia	No	417	52	366	-	--	--	17	11	120	--	13	37	226
	%	100.0	12.4	87.6	-	--	--	4.2	2.8	28.7	-	3.2	8.9	54.1
Headache	No	1,102	292	809	18	71	15	31	106	232	--	11	162	516
	%	100.0	26.5	73.5	1.6	6.4	1.3	2.8	9.6	21.1	--	1.0	14.7	46.9
Sight disorders	No	1,200	449	750	--	--	42	48	20	27	34	38	360	646
	%	100.0	37.5	62.5	-	-	3.5	4.0	1.7	2.2	2.9	3.1	30.0	53.8
Hearing disorders	No	1,028	607	422	24	28	48	56	22	18	18	18	527	335
	%	100.0	59.0	41.0	2.4	2.7	4.6	5.5	2.1	1.8	1.7	1.5	51.2	32.6
Hypertension	No	1,551	588	963	-	32	-	90	355	683	30	46	214	254
	%	100.0	37.9	62.1	--	2.0	-	5.8	22.9	44.1	1.9	3.0	13.8	16.4
Heart disease	No	847	429	418	55	50	52	42	240	234	207	141	170	175
	%	100.0	50.6	49.4	6.5	5.9	6.1	5.0	28.4	27.7	24.5	16.7	14.1	14.8
Acute respiratory	No	781	355	426	238	257	106	168	93	117	--	--		
	%	100.0	45.4	54.6	30.5	33.0	13.6	21.5	11.9	15.0	--	--		
Influenza	No	680	296	384	250	326	90	112	30	69	--	--		
	%	100.0	43.6	56.4	36.8	48.0	13.2	16.5	4.4	10.1	-	--		
Bronchitis and emphysema	No	562	279	283	23	18	16	22	40	25	39	16	205	230
	%	100.0	49.6	50.4	4.1	3.1	2.9	3.9	7.2	4.4	7.0	2.9	36.5	40.9
Asthma	No	547	290	257	11	20	14	17	40	56	51	46	206	177
	%	100.0	53.1	46.9	2.1	3.6	2.6	3.1	7.4	10.2	9.3	8.4	37.7	32.3
Hay fever and other allergies	No	2,157	987	1,170	5	12	67	54	46	48	19	9	862	1,064
	%	100.0	45.8	54.2	.2	.5	3.1	2.5	2.1	2.2	.9	.4	40.0	49.3
Dental problems	No	1,697	739	958	18	25	143	213	14	13	-	0	577	730
	%	100.0	43.6	56.4	1.0	1.5	8.4	12.6	.8	.8	-	0	34.0	43.0
Gastric and duodenal ulcers	No	482	282	199	--	14	--	10	88	58	12	--	193	131
	%	100.0	58.6	41.4	--	2.9	--	2.1	14.3	12.1	2.5	--	40.0	27.1
Digestive disorders	No	887	286	401	43	52	47	47	188	305	31	16	--	18
	%	100.0	41.7	58.3	6.3	9.0	6.8	6.8	28.8	44.4	4.5	2.4	--	2.6
Skin disorders	No	2,064	756	1,308	--	17	45	100	292	480	--	--	430	774
	%	100.0	36.6	63.4	--	.8	2.2	4.9	14.1	22.3	--	--	20.8	37.5
Arthritis and rheumatism	No	2,440	844	1,596	22	69	22	44	126	303	69	189	664	1,187
	%	100.0	34.6	65.4	.9	2.8	.9	1.8	5.2	12.4	3.7	7.7	27.2	48.7
Limb and joint disorders	No	2,334	1,182	1,153	69	88	139	156	77	104	258	258	770	696
	%	100.0	50.6	49.4	3.0	3.8	6.0	6.7	3.3	4.5	11.0	11.0	33.0	29.8
Trauma	No	616	349	268	111	90	172	122	33	32	117	88	82	38
	%	100.0	56.6	43.4	17.9	14.6	28.0	19.8	5.3	5.2	19.0	15.9	8.5	6.3
Other	No	2,680	965	1,715	150	413	253	510	347	574	248	385	163	246
	%	100.0	35.5	64.5	6.0	15.5	9.5	19.2	13.0	25.4	9.3	14.9	6.1	9.2

(1) "Prevalence" refers to existing conditions reported at the time of the interview and therefore includes both acute and chronic conditions.
(2) The top portion of the table shows the proportion of the population experiencing health problems while the bottom shows the number of health problems reported classified by type of problem.

Source: The Health of Canadians, Health and Welfare Canada (1981)
Table 60

The relationship between a disability day and lost time at work cannot be ascertained directly from the Canada Health Survey. What is required is an estimate of the average time spent at work (by occupation, age and sex). No single source provides all of this information but it is possible to assemble it from the Statistics Canada labour force surveys and census data.

Lost Time in Household Work

Methodologies for estimating the value of household work have been carefully reviewed by Hawrylyshyn (1978). He discusses three approaches:

i) the opportunity cost method (household work is valued at the average wage of similar people, by age and sex, in the labour force)

ii) the individual function cost method (household work is valued at the market wage paid for each function if performed by a professional)

iii) the housekeeper cost method (household work is valued at the market wage paid to a professional housekeeper)

He argues that the individual function cost method is to be preferred on theoretical grounds but notes that the opportunity cost method provides a low-cost first approximation estimate that is reasonably close to that of the preferred formula.

The valuation of household work by the individual function cost method requires information on time spent in the various household activities. Some data are available from the 1981 Canadian Time Use Pilot Study. This study is based on 24-hour time budgets reported by approximately 2,700 Canadians in the fall of 1981. While not strictly random, the sample does give a preliminary indication of how Canadians spend their time, and, in particular, can be used for estimating time spent in housework, child care, and shopping by age and sex.

Combining these estimates with those from the Canada Health Survey, and valuing the lost time in household work by one of the three methods noted earlier, will yield an indication of the value of household work lost because of ill health.

There is one remaining component for estimating the value of the health benefits from more stringent automotive emission controls. That is an estimate of the quantitative relation between emission controls and health impacts. In the absence of this link it is impossible to estimate the value of the health benefits that might be forthcoming from a reduction in automotive emissions. However, once such estimates are provided, the approach outlined in this section can be used to derive output accounting values (and lower bound welfare accounting values) for the resulting health benefits.

3. AGRICULTURAL CROPS

Two crop-growing areas in Canada where mean, growing-season ozone concentrations are above the threshold for agricultural crop damage have been identified by Environment Canada (Dann 1984). They are:

- The southwestern portion of Ontario and Quebec along the Windsor to Quebec City corridor, and
- Vancouver and the lower Fraser River valley.

In the provinces of Nova Scotia and New Brunswick mean, growing-season ozone concentrations are close to, but below, the threshold level where damage to agricultural crops can be observed, based upon limited monitoring data (Dann 1984).

The situation in other crop-growing areas of Canada is uncertain, since little monitoring of ambient ozone concentrations in those areas has been carried out.

3.1 Estimating the Value of Increased Crop Production in Ontario

A key step in estimating the value of increased production of agricultural crops would be to determine the extent to which the mean, growing-season ozone concentration would fall if the proposed light duty vehicle emission standards are implemented. This step has not been carried out, apparently because of the lack of detailed emission inventories and the limitations of current photochemical models.

It is not possible to determine the value of increased crop production that could be attributed to reductions in light duty vehicle emissions, but estimates of the value of aggregate crop losses in Ontario due to ozone damage are available. Linzon et al (1984) used a 7-step procedure to estimate the value of crop production increases that would be expected if ambient ozone concentrations did not exceed damage-causing levels. A summary of the 7 steps is as follows:

1. Determine the locations and the yield loss of various crops that are exposed to damage-causing levels of O_3 (e.g., above 0.03 ppm 7-hr. seasonal mean).

2. Identify any mitigative actions such as resistant cultivars and antioxidant chemicals that have been implemented to offset damages which impose extra costs on producers.

3. Based on dose-response information, calculate the total amount of production increases that could result from a lowering of O_3 concentrations.

4. To obtain an estimate of the monetary value of the production change, multiply the amount of change by the appropriate market price. However, if production increases or decreases are large relative to the entire market for the product in question, then the market prices of the crop could decrease or increase and one should use the new price to estimate the monetary value of the change in production.

5. To obtain the economic benefit to producers (farmers) of the reduction in O_3 levels, multiply the change in crop production by the appropriate price and subtract any extra costs that might be incurred in growing, harvesting, or transporting this extra production. This profit to farmers is a part of the benefit of the oxidant control.

6. Add to this profit any decrease in costs that might result if cultural practices currently being taken by growers (extra fertility, sprays, etc.) can be reduced or eliminated.

7. If prices of the crops are lowered because of the production increase (or raised because of production losses), consumers will also experience a net gain or loss in terms of their willingess to pay. This quantity must be calculated by obtaining estimates of the demand functions for each crop. Demand functions indicate the amounts of money that people are willing to pay (per unit of product) at different levels of consumption. If prices decline, many people are then able to pay less than they would be willing to pay for the products in question, and so they experience a monetary benefit or gain. The benefit consumers would experience (or the cost they would incur if prices were increased) is called Consumer's Surplus.

This procedure, or a variant of it, could be used to calculate the value of increased crop production due to implementation of the proposed light duty vehicle emission standards. In step 3, the amount by which O_3 concentrations are lowered would be the amount that could be attributed to decreases in light duty vehicle NOx and HC emissions.

The results of the Linzon et al (1984) study are summarized in Table 3.1. For each sensitive crop species, estimates of average crop loss and a range of plus or minus about 50 percent of the average were used to calculate the production increases that could result from a lowering of O_3 concentrations. The value of the increased yield of crops in Ontario if ozone damage was eliminated was estimated to be about $15 million per year. The range of values using the low and high estimates of losses due to ozone is $9 million to $23 million.

Assuming that a reduction in light duty vehicle emissions reduces ambient ozone concentrations in the crop-growing areas where ozone damage is currently being observed, a portion of the $15 million in increased crop production would be realized if the proposed light duty vehicle emission standards are implemented. Unfortunately, at this time, there is no basis for estimating the size of that portion.

3.2 Other Crop-Growing Areas in Canada

The area planted, production, and the value of production of agricultural crops grown in the other areas where ozone damage is a factor--Quebec and the lower Fraser River valley--are shown on Table 3.2 and Table 3.3 respectively.

Table 3.1

Estimated Crop Production Increases Due to Ozone Reduction in Ontario

Crop (1978-80 average price)	Region 5: Average Seasonal 7-Hr. Mean O_3 of 0.05 + 0.005 ppm					Region 4: Average Seasonal 7-Hr. Mean O_3 of 0.04 to .005 ppm				
	1978-80 average crop production (tonnes)	% loss due to ozone damage		Average potential production increase (tonnes)	$ value of production increase	1978-80 Average crop production (tonnes)	% loss due to ozone damage		Average potential production increase (tonnes)	$ value of production increase
		Average	Range				Average	Range		
White Bean ($376.42/tonne)	14 346	12	(6-18)	1 957	736 654	51 892	7	(4-11)	3 907	1 470 673
Potato ($110.13/tonne)	73 036	8	(4-12)	6 354	699 766	297 775	5	(3-8)	15 663	1 724 966
Onion ($149.78/tonne)	27 038	8	(4-12)	2 352	352 283	49 316	5	(3-8)	2 594	388 529
Sweet Corn ($290.27/tonne)	5 596	8	(4-12)	487	141 361	5 944	5	(3-8)	313	90 854
Lettuce ($317.46/tonne)	1 436	8	(4-12)	125	39 683	11 097	5	(3-8)	584	185 397
Radish ($460.35/tonne)	4 484	8	(4-12	390	179 537	718	5	(3-8)	38	17 493
Spinach ($436.21/tonne)	590	8	(4-12)	51	22 373	1 890	5	(3-8)	99	43 185

able 3.1, continued

Estimated Crop Production Increases Due to Ozone Reduction in Ontario

rop	Region 5: Average Seasonal 7-Hr. Mean O_3 of 0.05 + 0.005 ppm					Region 4: Average Seasonal 7-Hr. Mean O_3 of 0.04 to .005 ppm				
1978-80 verage price)	1978-80 average crop production (tonnes)	% loss due to ozone damage		Average potential production increase (tonnes)	$ value of production increase	1978-80 average crop production (tonnes)	% loss due to ozone damage		Average potential production increase (tonnes)	$ value of production increase
		Average	Range				Average	Range		
utabaga $101.32/tonne)	6 680	8	(4-12)	581	58 867	50 703	5	(3-8)	2 667	270 220
omato $359.03/tonne)	10 104	5	(3-8)	531	190 645	10 668	2	(1-3)	218	78 269
ucumber $235.68/tonne)	2 347	5	(3-8)	123	28 989	1 940	2	(1-3)	40	9 427
reen Bean $522.03/tonne)	577	5	(3-8)	30	15 661	3 531	2	(1-3)	72	37 586
oybean $210.65/tonne)	737 073	3	(2-4)	22 766	4 797 764	84 877	none			
rape $292.95/tonne)	997	3	(2-4)	31	9 081	63 060	none			
heat $98.52/tonne)	461 497	3	(2-4)	14 260	1 404 895	331 462	none			
obacco $2 825.99/ onne)	68 722	1	(0.5-1.5)	694	1 961 237	20 466	0.6	(0.3-0.9)	123	347 597
		Total			$10 638 796			Total		$4 664 196

Source: Linzon et al (1984)

Table 3.2

CROPS AT RISK OF OZONE DAMAGE
QUEBEC, 1981

	Area	Production	Value of Production
	(hectares)	(tonnes)	(000$)
Wheat	40 904	119 506	19 716
Grain Corn	165 446	975 436	143 232
Soybeans	3 555	7 173	1 845
Fodder Corn	84 391	2 877 000	71 918
Hay	965 455	4 935 000	227 011
Tobacco	3 568	7 238	23 471
Potatoes	17 172	341 800	40 170
Dry Field Beans	456	400	412
Onions	1 036	36 269	7 865
Tomatoes	1 517	13 119	3 698
Sweet Corn	9 343	56 717	8 260
Lettuce	1 150	26 249	6 564
Rutabagas	877	21 276	2 783
Beans	3 964	17 988	4 071
Cucumbers	1 145	19 170	3 982
Radishes	335	2 150	1 062
Spinach	152	820	301
Totals	1 300 466	9 457 311	566 361

Source: Special tabulation provided by the Agricultural Statistics Division of Statistics Canada.

Table 3.3

CROPS AT RISK OF OZONE DAMAGE IN CENSUS DIVISIONS 11, 13, 15 AND 9B* OF BRITISH COLUMBIA, 1981

	Area (hectares)	Production (tonnes)	Value of Production (000$)
Wheat	913	1 854	336
Potatoes	2 303	49 056	9 074
Silage Corn	7 462	191 812	4 916
Hay	25 868	161 753	14 558
Onions	140	4 165	1 569
Sweet Corn	1 126	10 838	2 237
Rutabagas	68	1 475	508
Lettuce	223	5 031	2 115
Spinach	11	122	105
Wax, Green, Snap Beans	629	4 191	1 173
Cucumbers	85	829	745
Grapes	32	289	149
Totals	38 860	431 415	37 485

* These census divisions include the lower Fraser River valley.

Source: Special tabulation provided by the Agricultural Statistics Division of Statistics Canada.

Estimates of total crop losses due to ozone such as the one discussed in section 3.1 for Ontario were not available for Quebec and the lower Fraser River valley.

As with Ontario, there is no basis for determining the portion of ozone damage that would be eliminated in Quebec and the lower Fraser River valley if the proposed light duty vehicle emissions standards were implemented.

APPENDIX 1

Evaluation of Visibility

Reproduced from:

Impact Assessment Work Group 1, "United States-Canada Memorandum of Intent on Transboundary Air Pollution", January 1983.

5.2.2 Evaluation of Visibility

Visibility impairment may adversely affect public welfare in essentially two areas: (1) the subjective enjoyment of the environment (aesthetics, personal comfort and well-being), and (2) transportation operations. The aesthetic aspects of visibility values can be categorized according to: (1) social/political criteria, community opinions and attitudes held in common about visibility; (2) economic criteria, the dollar cost/benefit associated with visibility; and (3) psychological criteria, the individual needs and benefits resulting from visibility. These categories are not exclusive, but relate to different approaches for measuring somewhat intangible values. Evidence on visibility effects is drawn from studies of social perception and awareness of air pollution, economic studies, and visibility/air transportation requirements. These studies are summarized in Table 5-3 and evaluated briefly below.

5.2.2.1 Aesthetic Effects

Assessment of the social, economic and psychological value of various levels of visibility is difficult. The criteria document, an EPA report to Congress (USEPA 1979), Rowe and Chestnut (1981), and Fox et al. (1979), discusses and evaluates several approaches that have been used or proposed towards this end. In particular, preliminary studies of social awareness/perception and the economic value of visibility in urban and nonurban areas support the notion that visibility is an important value in both settings.

Early social awareness studies (DHEW 1969; Schusky 1966; Wall 1973) conducted in polluted urban areas have generally shown that at higher pollution levels an increasing portion of the population is aware of air pollution and considers it a nuisance. In St. Louis, a linear relationship was observed between annual particle levels (50-200 $\mu g/m^3$ TSP) and public awareness and concern. At 80 $\mu g/m^3$ annual geometric mean TSP, about 10% of those surveyed reported air pollution as a nuisance (Schusky 1966). Although it is reasonable to attribute more of these and other perception results to particulate matter than to gaseous pollutants (Barker 1976; Wall 1973), the relative importance of visibility degradation by plumes and haze as compared to dustfall was not clearly addressed in these studies. A more recent study of perception of air pollution in Los Angeles (Flachsbart and Phillips 1980) represents the most comprehensive evaluation of major pollutant indicators and perception to date. Five gaseous pollutants (O_3, CO, NO_2, NO, SO_2), four particle related indices (TSP, dustfall, CoH, visibility) and six averaging times were compared with perceived air quality as reported by 475 respondents living in 22 residential areas in Los Angeles County. Only two indices, visibility and ozone, were consistently significantly related ($\alpha = 0.001$) to perceived air quality for all averaging times. The highest correlation coefficient (Kendall's τ) occurred for yearly visibility ($\tau = -0.29$) and for number of days

TABLE 5-3. SUMMARY OF QUALITATIVE EVIDENCE FOR VISIBILITY RELATED VALUES

	Effect of Increased Visibility	Affected Groups	Averaging Times[a]	Supporting Observation
Transportation	More efficient, lower risk operations, visual approach permitted	Airport users, operators civilian and military	1 - 3 hr readings	Visual approach permitted when visibility 3-5 miles; airport specific (FAA 1980b)
	Increased opportunity to operate aircraft	General aviation aircraft (no-instrument capable pilots, aircraft)	1 - 3 hr readings	VFR permitted when visibility 3 miles (FAA 1980a)
Aesthetic:				
1) Social Criteria	Decreased perception of air pollution	Substantial percentage of general population; urban areas	Daily to annual	Perception of air pollution in Los Angeles significantly related to visibility for all averaging times (Fischbert and Phillips 1980). Perception, annoyance significantly related to particulate matter (Schusky 1966)
	Options values; maintaining or increasing opportunity to visit less impaired natural and urban settings	Outdoor recreationists, campers, tourists	Daily, peak visitation in summer months	Aggregate of activity values in iterative bidding studies suggests importance of options values (Rowe and Chestnut 1981)
	Improved view of night sky	Amateur astronomers, other star watchers	Nightly	Decrease in star brightness by by fine particles (Leonard et al. 1977)
2) Economic Criteria	Increased property values	Home owners	Long-term	Property values related to perception of air pollution, hence visibility (Brookshire et al. 1979; Rowe and Chestnut 1981)
	Enhanced enjoyment (user or activity values) of environment in:			
	a) Urban settings	Urban dwellers	Long-term	Willingness to pay for increased visibility in urban (Brookshire et al. 1979) and nonurban settings (Rowe et al. 1980)
	b) Natural settings	Outdoor recreationists, campers, residents of non-urban areas	Daily, peak visitation in summer months	
3) Psychological Criteria	Existence values; maintaining pristine environments	General population	Long-term	Existence values may far outweigh activity or user values (Rowe and Chestnut 1981)
	Less concern over perceived health effects	General population urban areas	Daily to long-term	About 2/3 of bid for improved visibility in Los Angeles was related to concern over potential health effects (Brookshire et al. 1979)

[a] Represents staff judgement of most important averaging time based on supporting observation. Because averaging times are related it is difficult to specify single (long or short) averaging time as most significant. Perception of visibility is essentially instantaneous.

visibility was less than 3 miles (T = 0.32).[a] Of the other particle indices, only monthly average dustfall was significantly correlated (T = 0.12) with perception. Consistent with other studies, the survey also found that air quality is valued less by minority groups and long-time urban residents than by whites (all income classes) and those with some history of rural residence.

The two major approaches to economic valuation of visibility include: (1) survey (e.g., iterative bidding), and (2) property value studies. The major published iterative bidding studies of visibility, conducted in the rural southwest and in the Los Angeles area (South Coast Air Basin), are summarized and evaluated in Table 5-4. The Four Corners and Lake Powell studies deal only with single sources and visible plumes, while the Farmington and Los Angeles studies address haze. The preliminary nature of these studies makes them useful primarily as qualitative indicators of the economic value of visibility. Among the more important limitations of the published results are the following:

1. None of these studies has measured existence values (benefit of just knowing pristine areas exist, regardless of intent to use them) or options values (wish to preserve the opportunity to view an unimpaired vista). Rowe and Chestnut (1981) suggest that existence values of good visibility in natural settings may be significantly greater than measured activity values.

2. The studies may be subject in varying degrees to methodological problems. The Farmington study, in particular, discovered a number of biases probably related to the credibility of the contingent market. These biases were not always large but show the difficulty of valuing visibility through iterative bidding.

3. Even if the available results could be taken at face value, so few studies have been conducted that results cannot be directly transferred to other areas of the country. For example, it might be expected that willingness to pay for improved visibility in Los Angeles might be greater than that for areas in flat terrain without background hills or mountains.

Despite their limitations, the iterative bidding studies suggest that visibility is of substantial economic value in both urban and natural settings. Although the value of visibility in other areas may vary significantly from that suggested by studies in the rural southwest and Los Angeles, no a priori reason exists to suggest that visibility is of little value in heavily populated eastern urban and rural areas. With respect to recreational settings, of the 23 most heavily used national parks and monuments, 11 were in the East (NPS 1981). In 1979 over 90 million visits were recorded in all eastern National Park Service managed facilities.

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[a] Kendall's T is a nonparametric statistic. The negative correlation between the number of people perceiving poorer air quality and visibility and the positive correlation with the number of days visibility is less than 3 miles are consistent with expectations.

TABLE 5-4. SUMMARY RESULTS OF ITERATIVE BIDDING VISIBILITY STUDIES (after Rowe and Chestnut 1982)

Design Elements/Study (Year)	Four Corners NM (1972)[b]	Lake Powell UT/AZ (1975)[c]	Farmington NM (1977)[d]	South Coast Air Basin CA (1978)[e]
Total Interviews Conducted (Percents Usable)	1,099 (69%)	104 (79%)	130 (92%)	345 (NR)
Scenarios	Emissions, strip minings, transmission lines, single power plant	Air Quality and Power Plant	Air Quality and Power Plant	Air Quality and Health
	A-worst	A-No plant	A-Visibility 120 km	A-Poor Visibility-2 miles
	B-middle	B-Plant, no plume	B-Visibility 80 km	B-Fair Visibility-12 miles
	C-best	C-Plant with plume	C-Visibility 44 km	C-Good Visibility-28 miles
Payment Vehicle	User fees/ Electricity Bill	User Fees	Utility Bill/ Payroll Reduction User Fee	Utility Bill/ Monthly Payments to Conservation Fund
Willingness to Pay Bid Comparisons				
a) Yearly bids for individual residents households	A-C[a] $85 B-C $50 A-B -	--	A-D[a] $82 A-B $57 B-D $43	A-C[a] $245 B-C $243 A-B $174
b) Daily bid for individual recreationists min. $1.00	--	A-C $2.95	A-D $2.44	--
c) Aggregate Yearly Value	A-B $15.54 M A-C $24.57 M	A-B $.414 M A-C $.727 M	A-B $1.47 M A-C $1.99 M A-D $2.14 M B-C $1.1 M	30% Improvement $3.58 - $3.65 B
Tested for bias/found biases	No/No	No/No	Yes/Yes	Yes/Yes
Comment on Total Values	Values for total affected four corners region; attributed mostly to particulate air pollution reduction at single source, but difficult to separate from other visible factors due to unstandardized scenarios. Measured activity values only.	Examined one of fifteen potentially affected parks; pictoral representations not consistent across A-C. Measured activity values only.	Value for San Juan County, New Mexico and Navajo recreation area only. Affected area up to 10 times larger strategic bias not found, but other bias problems with contingent market found. Measured activity values only.	About one-third of benefits are related to aesthetics, two-thirds to health. Results reasonably consistent with companion property value study. Measured user values. Some questionnaire design biases related to aesthetics.

[a] Capital letters refer to scenarios listed above. Thus "A-C" in the Four Corners case refers to the bids for going from scenario A (worst) to scenario C (best).
[b] Randall et al. 1974
[c] Brookshire et at. 1976
[d] Rowe et al. 1980
[e] Brookshire et al. 1979

A large number of property value studies related to air quality have been conducted. These have been reviewed by Freeman (1979a,b) and Rowe and Chestnut (1981). Although a variety of air quality indicators have been used, the results of awareness/perception studies strongly suggest visibility plays an important role in air quality related impacts on property values (Rowe and Chestnut 1981). This contention is best documented in the case of the South Coast Air Basin property value survey (Brookshire et al. 1979). There, the estimated annual benefit of a 25-30% improvement in air quality based on property values was about $500 U.S. per household. These results are qualitatively similar to the companion iterative bidding study (about $300 U.S./household). The bidding study suggests that 22-55% of the bids to improve visibility were related to aesthetic effects. Both the bidding results and the perception study (Flachsbart and Phillips 1980) conducted in the same area support the possibility of substantial impacts of visibility on Los Angeles area property values.

None of the other published property value studies are accompanied by companion studies that suggest what portion willingness to pay for improved air quality may be due to visibility. Moreover, theoretical problems remain in relating willingness to pay functions from property value differentials (Rowe and Chestnut 1981). No single study has examined all of the variables that might be important in influencing property values. Because air pollution tends to be a small influence compared to other variables, earlier studies that examined a limited number of variables are particularly suspect. In essence, the available literature suggests that perceived air pollution, and hence visibility, may have tangible effects on property values in urban areas such as Washington, Boston, Los Angeles, and Denver (Rowe and Chestnut 1981); nevertheless, additional theoretical and empirical work is needed before reliable and transferable quantitative relationships for visibility evaluation can be established.

5.2.2.2 Transportation Effects

Although all forms of transport may be affected by poor visibility (e.g., slowing of highway traffic by anthropogenically induced fog), at current ambient levels, aircraft operations appear to be most sensitive. When visibility drops below 3 miles, both U.S. FAA and Canadian safety regulations restrict flight in controlled air spaces to those aircraft and pilots that are certified for operation under Instrument Flight Rules (IFR) (FAA 1980a). The most severe impact in such cases is usually on non-IFR general aviation aircraft which are, in effect, grounded or forced to search for alternate landing sites. In 1979, there were about 210,000 active general aviation aircraft which accounted for about 84% of total airport operations in the U.S. Over 23% of all general aviation aircraft had no IFR capability (Schwenk 1981). Estimates of the percentage of pilots certified for IFR in the U.S. and Canada are not available. Commercial, military, and other IFR aircraft operation also may be affected by reduced

visibility. Under IFR conditions, the number of arrivals and departures per hour can be significantly decreased as compared to Visual Flight Rules (VFR) conditions. The effect varies with airport, and in some cases, the visual range cutoff for the most efficient visual approaches (VAPs) may be 5 miles (FAA 1980b). For example, the performance standard for one configuration at Boston Logan International Airport is 109 operations per hour for VAPs (5 miles), 79 operations per hour for "basic" VFR (3-5 miles), 79 operations per hour for "controllers" visual approach IFR (2-3 miles), and 60 operations per hour for "category I" IFR (2 miles). Thus, depending on airport configuration, schedules, and the extent and duration of haze induced visibility reduction, delays in commercial and other aircraft operations can occur. In large segments of the eastern U.S., midday visibilities less than 3 miles with no obvious natural causes occur 2-12% of the days in the summer and 1-5% of the time during other seasons (USEPA 1981). Visibilities less than 5 miles would, of course, be more frequent. Based on typical eastern summertime diurnal cycles in humidity and light scattering (e.g., Ferman et al. 1981), the occurrence of morning visibilities (6-8 a.m.) less than 3-5 miles would be somewhat greater than for midday visibility, even discounting naturally occurring fog.

Compared with other modes of transport, air travel is generally considered to be safe. It is not, however, riskfree; based on reasonable expectations and the available record, air pollution visibility impairment would tend to increase risks of aircraft operation (U.S. Senate 1963). Failure to see and avoid objects and obstructions during flight is one of the ten most frequent cause factors for general aviation accidents (FAA 1978). Another important cause factor is continued VFR flying into adverse weather. Although such action is normally attributed to errors in judgment (FAA 1978), in some cases, pilots who by choice or necessity fly in the mixing layer, could fail to distinguish storm fronts or thunderclouds from the prevailing haze until they are upon the adverse weather. The available data in the criteria document do not, however, permit any quantitative assessment of the risks to commercial and general aviation aircraft operation associated with reduced visibility.

The available information of the effects of visibility on transportation suggests that episodic eastern regional haze tends to curtail substantial segments of general aviation aircraft and slow commercial, military, and other IFR operations on the order of 2-12% of the time during the summer. The extent of any delays varies with airport. Reduced visibility may also tend to increase risks associated with aircraft operations in the mixing layer, but quantitative assessments are not available.

5.2.3 Mechanisms and Quantitative Relationships

The mechanisms by which atmospheric pollutants degrade perceived visibility are reasonably well understood (Friedlander 1977;

APPENDIX 2

Diagnosis-Specific Inpatient Hospital Costs

Diagnosis-specific marginal costs (1969 dollars)

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
1	420.37	626.20	43.16	55.00
2	266.67	340.42	25.44	36.50
3	298.02	407.79	28.80	39.98
4	447.80	771.58	39.51	52.70
5	364.28	506.17	41.95	54.23
6	306.21	377.37	38.46	48.87
7	401.77	612.50	44.08	57.08
8	438.49	761.63	47.35	62.17
9	364.57	477.35	65.79	79.11
10	291.32	394.55	28.80	40.56
11	364.14	557.74	42.04	53.94
12	260.93	332.56	21.63	32.60
13	346.13	521.58	31.55	43.62
14	310.66	402.75	42.14	54.66
15	278.48	379.65	23.35	35.44
16	322.16	473.98	29.91	41.96
17	514.88	774.69	56.28	69.02
18	454.86	763.05	34.12	46.42
19	480.25	824.42	33.87	45.97
20	496.69	841.49	35.30	47.53
21	470.98	799.72	35.69	48.15
22	461.42	752.94	39.86	53.03
23	507.96	801.37	56.10	69.75
24	505.78	823.90	47.28	59.85
25	395.77	563.53	46.15	58.80
26	428.31	648.48	39.97	52.13
27	514.81	701.87	63.89	75.49
28	432.69	618.38	45.64	58.04
29	473.77	696.03	48.23	60.01
30	527.10	853.55	53.32	66.22
31	430.35	701.33	35.40	47.77
32	456.20	655.32	56.44	68.48
33	423.60	644.58	41.75	54.16
34	488.43	810.11	41.20	53.80
35	568.70	992.90	49.08	63.12

Key:

SSMCC1 = marginal inpatient cost per case, bed supply remaining constant

SSMCC2 = marginal inpatient cost per case, occupancy rate remaining constant

SSMCD1 = marginal inpatient cost per day, bed supply remaining constant

SSMCD2 = marginal inpatient cost per day, occupancy rate remaining constant

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
36	448.99	733.15	37.71	50.27
37	439.85	652.61	50.92	64.31
38	487.83	699.76	59.47	72.67
39	275.05	315.69	36.59	48.10
40	283.94	343.08	43.55	55.62
41	326.34	430.08	34.45	45.93
42	317.54	422.90	32.27	43.88
43	271.80	328.88	25.92	37.31
44	425.63	701.93	50.95	65.09
45	301.11	401.98	33.40	45.58
46	346.48	452.61	39.76	51.43
47	347.33	497.94	39.97	52.73
48	398.63	597.35	43.89	57.58
49	335.46	455.70	37.69	50.15
50	359.28	544.02	34.32	46.88
51	347.14	537.05	28.34	39.84
52	379.45	569.20	43.11	56.62
53	348.17	526.18	34.26	46.37
54	466.86	733.37	65.64	80.63
55	351.86	552.68	31.44	43.75
56	346.47	528.32	30.28	42.15
57	383.62	609.51	30.02	41.46
58	299.24	416.26	30.72	42.68
59	378.35	568.20	38.16	50.18
60	540.53	929.48	44.60	57.29
61	426.15	720.12	34.24	45.71
62	447.58	770.22	37.07	49.71
63	365.82	577.42	32.18	43.85
64	320.49	438.34	33.43	44.48
65	386.27	555.37	54.92	67.63
66	387.74	591.86	40.68	53.28
67	435.14	639.27	59.44	73.09
68	413.57	665.70	46.59	60.26
69	468.65	758.47	56.46	71.10
70	470.41	807.40	36.99	49.95
71	470.04	825.62	33.57	45.90

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
72	303.82	437.94	28.61	40.80
73	392.55	630.08	43.27	56.87
74	451.80	787.80	39.76	53.17
75	332.75	484.72	33.49	46.00
76	323.92	431.89	37.80	49.14
77	349.37	463.02	49.84	62.97
78	335.25	370.98	109.78	122.94
79	400.34	532.71	52.36	65.36
80	392.47	532.58	53.41	67.10
81	401.20	524.85	71.19	85.66
82	282.83	341.36	36.28	48.29
83	376.00	489.73	57.36	69.76
84	333.46	406.03	53.53	66.42
85	362.23	587.17	29.61	41.17
86	477.71	775.67	49.74	64.74
87	317.80	474.37	26.45	37.95
88	408.39	651.07	31.01	42.44
89	363.08	552.04	30.47	42.03
90	352.15	540.78	29.27	41.01
91	369.76	596.65	31.88	44.08
92	448.51	753.33	35.20	47.80
93	412.18	674.70	30.75	42.26
94	419.08	688.69	31.51	43.22
95	437.91	692.71	49.37	63.18
96	481.41	817.09	40.41	53.84
97	431.18	681.59	36.70	49.21
98	352.04	539.00	29.02	40.39
99	387.96	611.42	34.95	47.23
100	321.61	434.54	31.75	43.29
101	302.07	393.69	29.89	41.10
102	336.63	496.63	33.99	46.52
103	359.68	528.26	34.51	46.82
104	259.09	322.77	22.82	33.54
105	260.21	331.65	19.00	28.57
106	299.82	431.28	25.50	36.39
107	280.42	387.81	23.74	34.55
108	356.45	549.02	28.08	38.74
109	287.80	388.02	28.61	40.17
110	243.30	265.80	24.65	36.08

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
111	311.27	386.46	43.38	55.50
112	343.07	397.36	74.80	87.26
113	311.51	379.55	49.99	63.03
114	455.99	799.02	39.38	52.41
115	379.63	590.63	34.97	47.85
116	323.37	476.56	27.61	39.28
117	367.46	559.07	33.91	46.33
118	246.30	267.23	37.79	48.53
119	275.28	352.87	26.45	38.47
120	358.72	525.28	39.29	52.19
121	378.01	586.55	31.25	42.83
122	346.47	513.70	29.63	40.94
123	305.02	439.86	24.06	34.58
124	392.19	611.63	31.64	43.31
125	262.76	342.28	19.66	30.13
126	302.45	438.37	25.85	36.95
127	280.08	360.28	26.21	37.38
128	298.69	395.95	29.17	40.59
129	325.60	449.35	30.59	41.97
130	343.72	517.29	30.28	42.02
131	279.60	389.57	28.50	40.03
132	412.91	674.31	35.56	48.12
133	353.88	530.57	29.68	40.90
134	307.16	432.80	28.20	39.77
135	422.94	683.26	33.98	46.17
136	375.30	596.87	32.82	45.06
137	345.97	493.04	31.38	42.67
138	319.15	454.25	27.62	38.61
139	336.05	508.94	27.63	38.65
140	360.49	544.96	32.65	44.59
141	465.66	519.52	252.27	266.56
142	293.63	419.33	23.88	35.26
143	397.88	616.90	39.59	52.51
144	321.60	433.18	34.22	46.26
145	289.65	377.07	29.74	41.24
146	391.99	591.09	42.65	55.36
147	350.10	444.43	57.28	69.50
148	334.59	455.12	39.15	51.50
149	409.19	622.76	38.39	50.92

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
150	254.82	287.00	29.54	40.53
151	282.57	364.52	27.38	39.02
152	269.95	322.18	29.36	41.08
153	315.25	414.95	32.62	44.32
154	290.44	349.80	34.18	44.89
155	266.41	326.80	23.21	34.49
156	361.25	510.72	34.35	45.91
157	309.42	389.42	35.39	46.23
158	269.66	319.18	28.41	39.68
159	287.25	354.77	31.02	42.41
160	289.01	356.64	30.34	41.75
161	262.38	320.60	21.45	32.47
162	257.47	294.28	25.78	37.10
163	288.95	363.57	29.17	40.41
164	308.25	350.31	63.92	76.95
165	295.91	366.66	33.05	44.39
166	354.54	474.63	39.37	51.17
167	398.53	529.16	47.03	59.39
168	359.45	448.70	47.67	59.42
169	359.09	469.83	41.46	53.17
170	359.95	469.04	43.48	55.53
171	259.53	304.04	22.81	34.21
172	352.25	439.88	48.91	59.30
173	304.55	388.47	32.21	43.68
174	280.91	393.70	23.74	35.30
175	320.22	489.01	28.54	40.52
176	299.88	417.39	29.17	41.08
177	472.19	821.63	38.92	51.34
178	415.16	697.83	35.02	47.87
179	314.15	477.15	25.90	37.08
180	394.19	637.82	35.82	48.49
181	386.89	599.91	34.16	46.11
182	324.68	442.45	35.71	47.98
183	266.03	325.75	26.75	37.95
184	338.29	475.69	38.63	51.13
185	599.84	1062.55	75.49	90.62
186	522.11	844.33	67.87	82.71
187	403.25	520.64	90.56	104.92
188	333.38	387.87	83.00	96.17

Diagnostic category	SSMCC1	SSMCC2	SSMCD1	SSMCD2
189	520.23	765.92	81.20	97.32
190	585.30	897.56	82.92	99.79
191	502.91	756.77	82.74	98.89
192	444.34	614.84	80.09	94.98
193	490.19	645.72	105.39	120.86
194	456.27	628.85	83.08	98.04
195	380.14	579.05	47.06	59.89
196	343.14	463.64	45.34	58.18
197	367.45	483.46	55.96	69.00
198	425.74	629.09	54.55	68.47
199	346.86	487.29	47.46	60.62
200	321.75	467.30	27.93	41.23
201	382.55	490.08	54.66	66.78
202	427.21	628.22	55.63	68.99
203	393.80	520.68	54.14	66.92
204	490.67	746.27	57.53	71.00
205	419.92	533.56	60.14	72.59
206	274.47	334.14	36.41	48.64
207	292.78	366.26	36.25	48.47
208	360.66	532.26	47.28	60.67
209	316.04	416.36	38.97	51.47
210	316.38	426.26	38.72	51.27
211	384.20	624.27	30.03	41.42
212	259.53	330.97	20.78	31.94
213	552.93	995.17	34.27	46.37
214	333.08	496.25	28.79	40.18
215	270.79	367.94	21.68	32.92
216	255.07	315.52	21.67	33.09
217	476.10	817.33	55.47	70.18
218	344.59	514.65	31.14	42.84
219	229.73	284.14	7.87	18.53
220	309.32	401.03	38.90	51.13
221	267.44	319.65	32.07	43.12
222	416.11	712.79	34.75	46.59
223	249.89	314.88	17.62	28.62
224	241.42	306.57	14.09	24.58
225	265.99	314.14	33.99	46.17
226	362.47	589.13	30.22	41.92
227	352.56	533.61	41.44	54.35
228	271.04	339.00	26.02	37.66
229	236.01	282.82	13.22	24.51
230	266.06	362.60	19.34	30.26
231	324.80	462.89	34.46	47.20
232	305.55	395.40	33.25	45.46
233	361.71	413.13	136.32	149.63
234	289.64	336.02	41.34	53.13
235	293.88	363.68	32.76	44.15
236	410.86	606.95	34.31	45.66
237	376.27	568.77	32.69	49.03

Source: Barer (1981)

Definition of Diagnosis Category Code

Adjusted Ontario Broad Codes and case complexities

237-Category code	1969 OBCs	ICDA-8 Codes, 1969	Description
001	001	000-003	Salmonella infections, cholera, and typhoid
002	002	004-009.0 009.2, 009.9	Other intestinal infections
003	003	009.1	Diarrhea
004	004	010-019	Tuberculosis
005	005, 006	020-027, 030-033, 039	Other bacterial diseases
006	007	034, 035	Streptococcal sore throat, scarlet fever, and erysipelas
007	009	038	Septicemia
008	012	044	Late effects of acute poliomyelitis
009	010, 011, 013	045, 046, 040-042, 043	Aseptic meningitis and other enterovirus diseases of central nervous system; poliomyelitis
010	014	050-057	Viral diseases accompanied by exanthem
011	015	062-066	Viral encephalitis
012	016	071-079, 060-061, 067-068	Other virus diseases
013	017	070	Infectious hepatitis
014	018	090-099	Venereal diseases
015	019	100-104, 080-089	Other spirochetal diseases
016	020	110-117, 120-129, 130-136	Mycoses, helminthiases, and other infective and parasitic diseases
017	021	140-149	Malignant neoplasm of buccal cavity and pharynx
018	022	151	Malignant neoplasm of stomach

237-Category code	1969 OBCs	ICDA-8 Codes, 1969	Description
019	023, 024	152, 153	Malignant neoplasm of small intestine, including duodenum Malignant neoplasm of large intestine except rectum
020	025	154	Malignant neoplasm of rectum and rectosigmoid junction
021	026	150, 155-159	Malignant neoplasm of other digestive organs
022	028	162.1	Malignant neoplasm of bronchus and lung
023	027, 029	160-161, 162.0, 163	Malignant neoplasm of trachea Malignant neoplasm of other respiratory organs
024	030	170	Malignant neoplasm of bone
025	031	172, 173	Malignant neoplasm of skin
026	032	174	Malignant neoplasm of breast
027	033	180	Malignant neoplasm of cervix uteri
028	034, 036	181, 182, 183.1, 183.9	Malignant neoplasm of uterus, fallopian tube, broad ligament
029	035	183.0	Malignant neoplasm of ovary
030	037	184	Malignant neoplasm of other and unspecified female genital organs
031	038	185	Malignant neoplasm of prostate
032	039	186, 187	Malignant neoplasm of other and unspecified male genital organs
033	040	188	Malignant neoplasm of bladder
034	041	189	Malignant neoplasm of other and unspecified urinary organs
035	042	191	Malignant neoplasm of brain

Key:

OBCs: Ontario Board Code

ICDA-8: International Classification of Disease - 8th Revision

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
036	043	171, 190, 192-199	Other primary and secondary malignant neoplasms
037	044	204-207	Leukemia
038	045	200-203, 208-209	Other neoplasms lymphatic and hematopoietic tissue
039	046	217	Benign neoplasm of breast
040	047	216	Benign neoplasm of skin
041	048	218-219	Benign neoplasm of uterus
042	049	220	Benign neoplasm of ovary
043	050	221	Benign neoplasm of other female genital organs
044	052	225	Benign neoplasm of brain and other parts of nervous system
045	051, 053	210-215, 223, 224, 226-228, 222	Benign neoplasm of male genital organs Other benign neoplasms
046	054	234.0	Carcinoma in situ of cervix uteri
047	055	230-233, 234.1, 234.9, 235-239	Other neoplasms of unspecified nature
048	056	242	Thyrotoxicosis with or without goiter
049	057	240, 241	Nontoxic goiter
050	058	243-246	Other diseases of thyroid gland
051	059	250	Diabetes mellitus
052	060	251-258	Other endocrine diseases
053	061, 062	268 260-267, 269	Nutritional marasmus, avitaminoses, and other nutritional deficiency
054	063	270-273	Congenital disorders of metabolism
055	064	274-279	Other metabolic diseases

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
056	065	280	Iron deficiency anaemias
057	066	281	Pernicious anaemia and other deficiency anaemias
058	067	282-289	Other diseases of blood and blood-forming organs
059	068	291	Alcoholic psychosis
060	069	295	Schizophrenia
061	070	296	Affective psychoses
062	071	290, 292-294, 297-299	Other psychoses
063	072	300	Neuroses
064	073	303	Alcoholism
065	074	304	Drug dependence
066	075	301-302, 305-309	Other non-psychotic mental disorders
067	076	310-315	Mental retardation
068	077, 008	320-324, 036, 037	Meningitis and other inflammatory diseases of the central nervous system
069	078, 079	330-332, 333	Hereditary and familial diseases of the nervous system
070	080	340	Multiple sclerosis
071	081	342	Paralysis agitans
072	082	345	Epilepsy
073	083	346-348	Migraine, other diseases of brain and motor neurone diseases
074	084	341, 343-344, 349	Other diseases of central nervous system
075	085	350-358	Diseases of nerves and peripheral ganglia
076	086	360	Conjunctivitis and ophthalmia

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
077	087	361-369	Inflammatory diseases of the eye
078	088	373	Strabismus
079	089	374	Cataract
080	090	375	Glaucoma
081	091	370-372, 376-379	Other diseases of the eye
082	092	381	Otitis media without mention of mastoiditis
083	093	382-383	Mastoiditis with or without otitis media
084	094	380, 384-389	Other diseases of ear and mastoid process
085	095	390-392	Active rheumatic fever
086	096	393-398	Chronic rheumatic heart disease
087	097	400-404	Hypertensive disease
088	098	410	Acute myocardial infarction
089	099	411-414	Other ischemic heart disease
090	100	420-429	Other forms of heart disease
091	101	431	Cerebral hemorrhage
092	102	432-434	Cerebral embolism and thrombosis
093	103	430, 435-438	Other cerebrovascular disease
094	104	440	Arteriosclerosis
095	105	441	Aortic aneurysm
096	106	442-448	Other diseases of arteries, arterioles, and capillaries
097	107	450	Pulmonary embolism and infarction
098	108	451	Phlebitis and thrombophlebitis
099	109	452, 453	Venous embolism and thrombosis
100	110	454	Varicose veins of lower extremities
101	111	455	Hemorrhoids
102	112	457	Non-infective disease of lymphatic channels

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
103	113	456, 458	Other diseases of the circulatory system
104	114	460-466	Acute upper respiratory infection, except influenza
105	115	470-474	Influenza
106	116	480-486	Pneumonia
107	117	490, 491	Bronchitis
108	118	492	Emphysema
109	119	493	Asthma
110	120	500	Hypertrophy of tonsils and adenoids
111	121	503	Chronic sinusitis
112	122	504	Deflected nasal septum
113	123	501, 502, 505-508	Other diseases of upper respiratory tract
114	124	510, 513	Empyema and abscess of lung
115	125	515, 516	Pneumoconiosis and related diseases
116	126	511, 512, 514	Other diseases of respiratory system
117	127	517-519	Other chronic interstitial pneumonia, bronchiectasis and all other diseases of respiratory system
118	128	520-525	Diseases of teeth and supporting structures
119	129	526-529	Other diseases of oral cavity, salivary glands, and jaws
120	130	530	Diseases of esophagus
121	131	531	Ulcer of stomach
122	132	532	Ulcer of duodenum
123	133	533	Peptic ulcer, site unspecified
124	134	534	Gastrojejunal ulcer
125	135	535	Gastritis and duodenitis

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
126	136	536, 537	Other diseases of stomach and duodenum
127	137	540-543	Appendicitis
128	138	550, 551	Hernia without mention of obstruction
129	139	552, 553	Hernia with obstruction
130	140	560	Intestinal obstruction without mention of hernia
131	141	561	Gastroenteritis and colitis, except ulcerative, of non-infectious origin
132	142	563	Chronic enteritis and ulcerative colitis
133	143	562	Diverticula of intestine
134	144	564-569	Other diseases of intestines and peritoneum
135	145	571	Cirrhosis of liver
136	146	570, 572, 573	Other diseases of liver
137	147	574	Cholelithiasis
138	148	575	Cholecystitis and cholangitis, without mention of calculus
139	149	576	Other diseases of gallbladder and biliary ducts
140	150	577	Diseases of pancreas
141	151	580-584	Nephritis and nephrosis
142	152	590	Infections of kidney
143	153	591	Hydronephrosis
144	154	592, 594	Calculus of urinary system
145	155	595	Cystitis
146	156	596	Other diseases of bladder
147	157	598	Stricture of urethra

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
148	158	593, 597, 599	Other diseases of urinary system
149	159	600	Hyperplasia of prostate
150	160	605	Redundant prepuce and phimosis
151	161	601-604, 606-607	Other diseases of male genital organs
152	162	610, 611	Diseases of breast
153	163	612-616	Diseases of ovary, fallopian tube, and parametrium
154	164	620	Infective diseases of cervix uteri
155	165	622	Infective diseases of uterus (except cervix), vagina, and vulva
156	166	623	Uterovaginal prolapse
157	167	624	Malposition of uterus
158	168	626	Disorders of menstruation
159	169	621, 625, 627-629	Other diseases of female genital organs
160	170	630, 635	Infection of genital tract during pregnancy and urinary infections during pregnancy and puerperium
161	171	632	Hemorrhage of pregnancy
162	172	631, 633, 634	Other complications of pregnancy
163	173, 215	636-639, 762	Pre-eclampsia, eclampsia, toxemia-unspecified, and hyperemesis gravidarum; Renal disease and other toxemias of pregnancy and the puerperium
164	175, 223	640-645, 773	Abortion, termination of pregnancy
165	176	650	Delivery without mention of complication

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
166	177	651-653	Delivery complicated by: placenta previa or antepartum hemorrhage, retained placenta or other post-partum hemorrhage
167	178, 179	654, 655	Delivery complicated by bony pelvis Delivery complicated by fetopelvic disproportion
168	180	656	Delivery complicated by malpresentation of fetus
169	181	657	Delivery complicated by prolonged labor or other origin
170	182	658-662	Delivery with other complications including anesthetic; death in uncomplicated delivery
171	183	670-678	Complications of puerperium
172	184	684	Impetigo
173	185	685	Pilonidal cyst
174	186	680-683, 686	Infections of skin and subcutaneous tissue
175	187	690-698	Other inflammatory conditions of skin and subcutaneous tissue
176	188	700-709	Other diseases of skin and subcutaneous tissue
177	189	712	Rheumatoid arthritis and allied conditions
178	190	713	Osteoarthritis and allied conditions
179	191	710, 711, 714-718	Other arthritis and rheumatism
180	192	720-723	Osteomyelitis and other diseases of bone
181	193	725	Displacement of intervertebral disc
182	194	724, 726-729	Other diseases of joint
183	195	731	Synovitis, bursitis, and tenosynovitis
184	196	730, 732-738	Other diseases of musculoskeletal system
185	197	741, 742	Spina bifida and congenital hydrocephalus

237-Category code	1969 OBCS	ICDA-8 Codes, 1969	Description
186	198	743	Other congenital anomalies of nervous system
187	199	744	Congenital anomalies of eye
188	200	745.0-745.3	Anomalies of ear
189	201	746	Congenital anomalies of heart
190	202	747.0-747.2	Patent ductus arteriosus, coarctation of aorta, and other anomalies of aorta
191	203	747.3-747.9	Other congenital anomalies of circulatory system
192	204, 205	748.1, 749.0	Other anomalies of nose, cleft palate
193	206	749.1	Cleft lip
194	207	749.2	Cleft palate with cleft lip
195	208	750, 751	Other congenital anomalies of digestive system
196	209	752, 753	Congenital anomalies of genito-urinary system
197	210	754	Clubfoot (congenital)
198	211	755, 756	Congenital anomalies of musculoskeletal system
199	212	740, 745.4-745.9 748.0, 748.2-748.6, 748.8, 749.9, 757-759	Other and unspecified congenital anomalies
200	213, 214, 216	760, 761, 763	Chronic circulatory and genitourinary diseases in mother; maternal ante- and intrapartum infection; other maternal conditions unrelated to pregnancy
201	217	764.0-764.3, 765.0-765.3, 766.0-766.3, 767.0-767.3, 768.0-768.3,	Birth injury

237-Category code	1969 OPCS	ICDA-8 Codes, 1969	Description
202	218	764.4, 765.4, 766.4, 767.4, 768.4, 776	Asphyxia, anoxia, or hypoxia
203	219, 221	774, 775, 778.2	Hemolytic or hemorrhagic disease of newborn
204	220	777	Immaturity unspecified (excludes immature newborn)
205	222	764.9, 765.9, 766.9, 767.9, 768.9, 778.0, 778.1, 778.3, 778.9, 769-771, 779	Other causes of perinatal morbidity and mortality
206	224	793	Observation, without need for further medical care
207	225	780-792, 794-796	Symptoms, senility, and ill-defined conditions
208	226	800, 801	Fracture of skull
209	227	802.2-802.4	Fracture of jaw
210	228	802.0, 802.1, 802.5, 802.9, 803, 804	Other fractures of skull and face bones
211	229	805-809	Fractures of spine and trunk
212	230	810-819	Fracture of upper limb
213	231	820-821	Fracture of femur
214	232	822-829	Other fractures of lower limbs
215	233	830-848	Dislocation without fracture, sprains of joints and adjacent muscles
216	234	850, 854	Intracranial injury excluding those with skull fracture
217	235, 236, 237	851.0, 852.0, 853.0 851.1, 852.1, 853.1, 851.9, 852.9, 853.9	Intracranial injury without mention of open intracranial wound; with open intracranial wound; late effects
218	238	860-869	Internal injury of chest, abdomen, and pelvis
219	239	873, 879.7-879.9	Other and unspecified laceration of head and other multiple and unspecified open wounds of head, neck and trunk without mention of complication, with complication and late effect
220	240	870-872	Open wound of eye and ear
221	241	885-887	Traumatic amputation of upper limbs
222	242	895-897	Traumatic amputation of lower limbs
223	243	874-879.1, 880-884, 890-894 900-918	Other lacerations and open wound of head, neck and trunk
224	244	920-929	Contusion and crushing with intact skin surface
225	245	930-939	Effects of foreign body entering through orifice
226	246	940-949	Burns
227	247	950-959	Injury to nerves and spinal cord
228	248	960-979	Adverse effects of medical agents
229	249	980-989	Toxic effect of substances chiefly non-medicinal as to source
230	250	990-996	Other adverse effects
231	251	997-999	Complications peculiar to certain surgical and medical procedures
232	254	Y10	Medical and surgical aftercare
233	255	Y11, Y12	Plastic surgical treatment and fitting of prosthetic device
234	252, 253, 256, 259	Y00.0-Y00.9, Y01, Y03-Y09, Y13	Medical or special examination, prophylatic inoculation and vaccination, special conditions and examinations without sickness, mature infant not classified as newborn
235	257	Y20, Y22, Y23, Y26, Y27	Mature newborn
236	258	Y21, Y24, Y25, Y28, Y29	Immature newborn
237	260	Y14	Donors for organ transplant surgery

Source: Barer (1981)

Estimates of the Direct Burden of Ill-Health, Ontario, 1971

Rank	Diagnostic Categories*	Hospital Services	Physicians' Services	Dental Services	Pharmaceuticals	Total	%
1	Digestive	$ 97,426,556	$ 28,057,240	$ 138,355,968	$ 13,900,764	$ 277,740,528	13.9
2	Mental	221,424,828	38,238,752		13,628,200	273,291,780	13.6
3	C.V.S.	176,797,403	41,035,184		41,157,164	258,989,751	12.9
4	Respiratory	65,934,840	57,304,594		46,063,317	169,302,751	8.5
5	Accidents, etc.	86,185,575	37,481,944		11,175,124	134,822,643	6.7
6	Genito-Urinary	62,558,543	37,258,111		17,989,224	117,805,878	5.9
7	Special	59,183,525	27,423,923†		29,709,476	116,316,924	5.8
8	Neoplasms	85,575,585	23,199,519		5,996,408	114,771,512	5.7
9	C.N.S., etc.	50,348,946	37,219,702		18,806,916	106,375,564	5.3
10	Musculoskeletal	54,106,109	24,254,891		13,355,636	91,716,636	4.6
11	Pregnance, etc.	71,116,097	14,268,906		2,725,640	88,110,643	4.4
12	Endocrine, etc.	28,089,881	14,536,044		27,528,964	70,154,889	3.5
13	Symptoms	18,998,420	30,054,747		10,357,432	59,410,599	3.0
14	Skin	12,828,900	17,721,999		11,175,124	41,726,023	2.1
15	Infective	21,565,856	12,005,926		5,996,408	39,568,190	2.0
16	Congenital	15,848,007	3,483,640		272,564	19,604,211	1.0
17	Blood	7,021,658	5,375,301		2,453,076	14,850,035	0.7
18	Perinatal	7,657,162	389,710		272,564	8,319,436	0.4
	Total	$1,142,667,891	$ 449,290,133	$ 138,355,968	$ 272,564,001	$2,002,877,993	100.0

* According to ICDA — 8th Revision
† Excludes examinations of mature and immature new born infants.

Sources:
Hospital Services: R. D. Fraser, and M. G. Prime, "The Direct Costs of Hospital and Related Services, Ontario, 1971," Table 7.
Physicians' Services: R. A. Spasoff, "Expenditures for Physicians' Services, by Disease Category, Ontario, 1971," Table 5.
Dental Services: Ontario Ministry of Health, "Direct Costs of Dental Services in Ontario, 1971," Summary Table, p. 13.
Drugs and Pharmaceuticals: Compiled from data found in "Drug Costs, 3rd Draft." The estimates embody revisions discussed.

Estimates of Indirect Burden, Ontario, 1971

Rank	Diagnostic Categories*	Morbidity (thousands)	%	Mortality at 7% (thousands)	%	Sub total indirect burden (thousands)	%
1	C.V.S.	$15,477.4	9.9	$248,741.9	30.4	$264,219.3	27.1
2	Accidents	8,873.2	5.7	245,112.0	30.0	253,985.2	26.1
3	neoplasms	6,774.1	4.3	156,345.8	19.1	163,119.9	16.8
4	Mental	67,905.6	43.6	7,402.7	0.9	75,308.3	7.7
5	Digestive	14,030.7	9.0	40,490.7	5.0	54,521.4	5.6
6	Respiratory	4,740.0	3.0	29,250.3	3.6	33,990.3	3.5
7	Perinatal	3.3	—	22,293.1	2.7	22,296.4	2.3
8	C.N.S., etc.	5,026.2	3.2	13,607.7	1.7	18,633.9	1.9
9	Congenital	534.1	0.3	15,762.6	1.9	16,296.7	1.7
10	Endocrine	2,541.2	1.6	13,483.3	1.7	16,024.5	1.7
11	Genito-Urinary	7,318.7	4.7	7,530.6	0.9	14,849.3	1.1
12	Musculoskeletal	7,530.3	4.8	2,673.0	0.3	10,203.3	1.1
13	Obstetrical	8.658.2	5.6	996.1	0.1	9,654.3	1.0
14	Infective	1,136.8	0.7	6,582.4	0.9	7,719.2	0.8
15	Ill-defined	1,865 1	1.2	5,357.8	0.7	7,222.9	0.7
16	Blood	392.3	0.3	1,749.9	0.2	2,142.2	0.2
17	Skin	1,486.9	1.0	559.0	0.1	2,045.9	0.2
18	Special	1,526.0	1.0	.0	.0	1,526.0	0.2
	Total	$155,820.1	100.0	$817,938.9	100.0	$973,759.0	100.0

* According to ICDA — 8th Revision.

Source:
Computer Printout entitled, "1971, Total Burden of Ill Health in Ontario: Mortality and Morbidity," Table 3B and Table 3A.

Source: Fraser, Spasoff and Prime (1976)

APPENDIX 3

Appendix 3

Notes on the Theory of Welfare Accounting

Further insight into the meaning of and relationship among the four welfare accounting measures mentioned in Section 2.3.1.2 can be obtained with the use of some simple welfare economics constructs.

Figure A1 shows indifference curves for an individual. Each curve represents various combinations of yearly incomes and mortality (or morbidity) rates among which the individual has no preference, i.e., he or she is indifferent. Curve U_1 corresponds to combinations of risk and income, all of which are preferred by the individual to the combination of curve U_0. (For any level of risk, the individual prefers more income to less; and, for any level of income, the individual prefers less risk to more). The shape of the curves reflects the assumption that, to maintain the same level of welfare, increasingly large increments of income are required to compensate the individual for equal increments in the level of risk.

In Figure A2 the initial level of environmental risk is R_1 and the individual's income is R_1A. Assume for the moment that no opportunities exist for the individual to change this combination of income and risk either through a change in employment conditions or purchases of risk reducing commodities.

Now consider the implications of a possible or actual increase in environmental risk, from R_1 to R_2 in Figure A2. The individual's welfare falls to U_0 with a combination of risk and income given by C.

This change in welfare can be represented by a change in income in two different ways:

In Figure A2 AB shows the maximum sum of money the individual could pay to avoid the increase in risk and be at least as well off as with the increase in risk.

CD shows the minimum sum of money required by the individual to

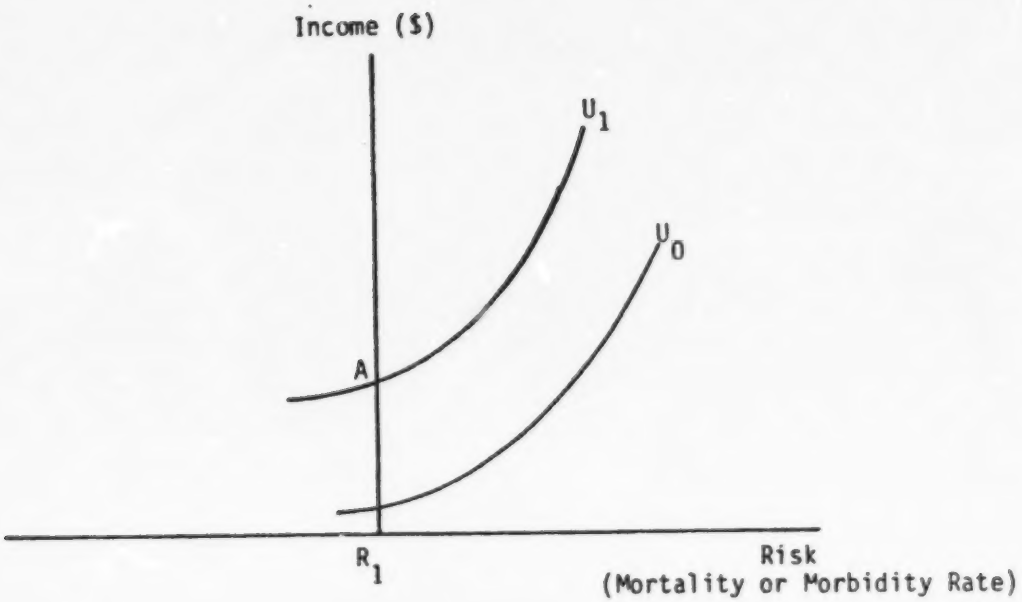

Figure A1

An Individual's Indifference Curves
for Income and Risk

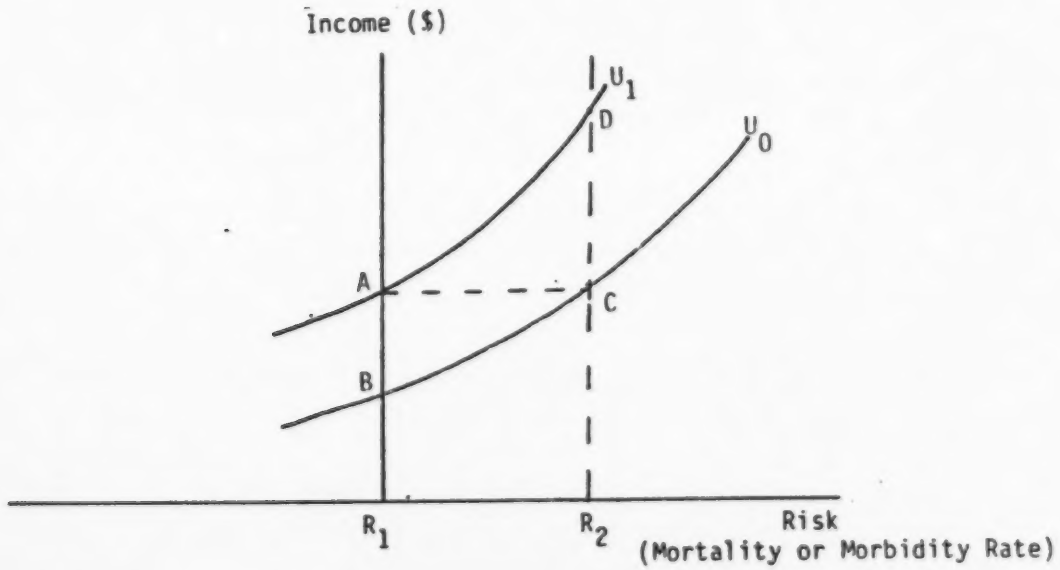

Figure A2

An Increase in Risk

fully compensate for the increase in risk.

There is no a priori reason to assume that AB = CD. Conventional assumptions about the individual's utility functions (in the case of risk aversion) imply that CD exceeds AB.

Figure A3 shows a similar analysis for a decrease in environmental risk from R_1 to R_0 where the negative change in risk is identical to the positive change in risk considered in Figure A2. In this case EF is the individual's maximum willingness to pay to receive the risk reduction and AG is the individual's minimum requirement for compensation to forego the reduction in risk. Again, there is no reason to assume that EF = AG. Conventional theory for risk averse individuals suggests that AG exceeds EF. Furthermore, in general, more of the four-value measures of the change in risk will be the same (i.e., AB = CD = EF = AG).

It should be noted that if the risk reduction had been from R_2 to R_1 instead of R_1 to R_0 then these would only be two-value measures instead of the above four. The willingness to pay for a reduction in risk from R_2 to R_1, AB, equals the compensation required to forego an increase in risk from R_1 to R_2. Likewise, the compensation required to accept an increase in risk from R_1 to R_2, CD, equals the willingness to pay to avoid a reduction in risk from R_2 to R_1.

These results draw attention to the relationship between the welfare accounting valuation of changes in risk and the initial level of risk. Also, the valuations will vary from person to person, for the same changes in risk. This tends to undermine attempts to generalize risk valuations from one person or group to another.

The next step in this analysis is to introduce the possibility of market transactions whereby an individual can substitute income for risk. This is shown in Figure A4.

The initial level of environmental risk is R_1 and the individual's income is R_1N with a welfare level of U_1. By increasing additional risk, the

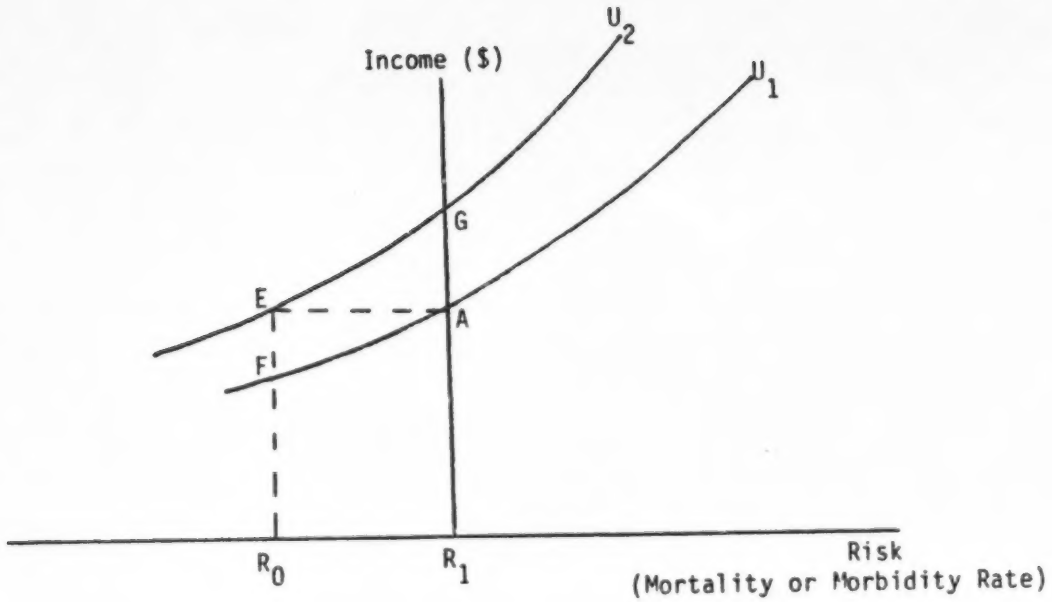

Figure 9 A3

A Reduction in Risk

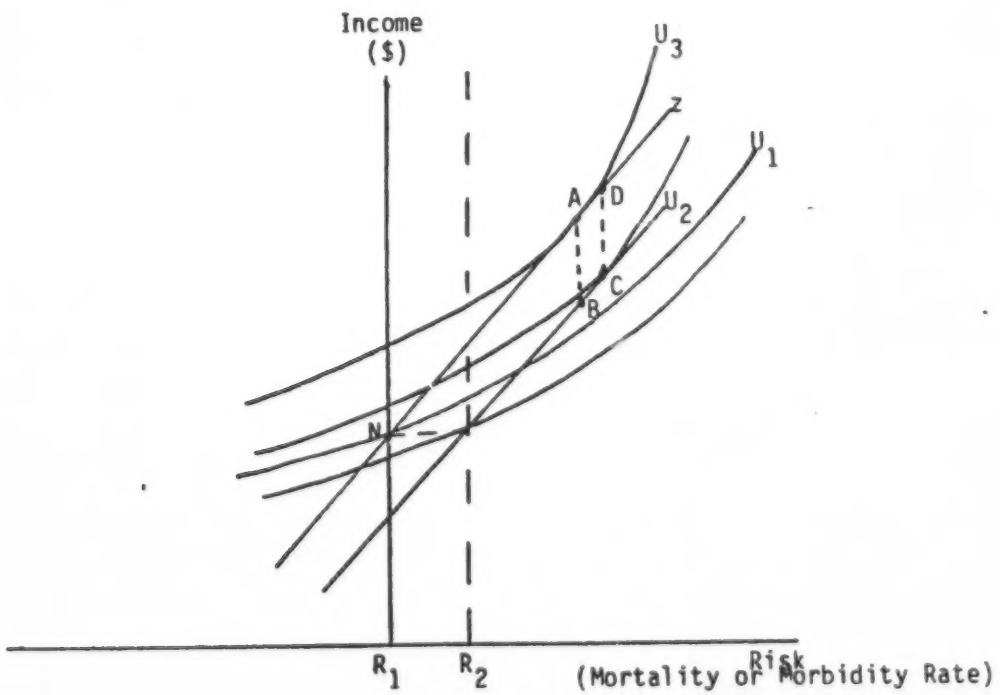

Figure 10 A4

**An Increase in Risk
and Voluntary Adjustment**

individual can obtain an increase in income according to the line Nz, the slope of which equals the rate of risk compensation in the labour market. This allows the individual to obtain a welfare level of U_3 at point A. Now consider the valuation of an increase in environmental risk from R_1 to R_2.

The individual would move to point C, at a lower welfare level, U_2. Rather than incur this increase in environmental risk, the individual would pay a maximum of AB, voluntarily incur more risk and income, and obtain point C. Minimum compensation required by the individual to incur the increase in risk is CD. Unlike the previous case, AB = CD.

A similar equality between willingness to pay and willingness to accept compensation applies in the case of a decrease in environmental risk. If the same starting point is adopted, i.e., environmental risk level R_1, then two different values for increases and decreases in risk will be obtained. But, if the increase and decrease in risk are merely reversals of the same situation, then all four welfare measures of the value of the change in risk will be identical.

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